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CLIMATE AND FEVERS OF INDIA

' Quale colui ch' è sì presso al riprezzo
Della quartana c' ha già l' unghie smorte,
E triema tutto, pur guardando il rezzo ;'

DANTE, *Inferno*. Canto decimosettimo.

ON THE
CLIMATE AND FEVERS OF INDIA

BEING

The Groonian Lectures

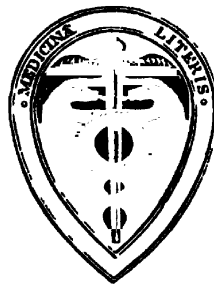
DELIVERED AT THE ROYAL COLLEGE OF PHYSICIANS

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BY

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PREFACE.

THESE LECTURES were delivered before the President and Fellows of the Royal College of Physicians in March 1882, and were published in the Medical Journals. They are now republished, with several additions and a series of Cases which illustrate the most important features of the diseases described.

I desire to return sincere thanks to my Brother Officers and friends for many valuable contributions which appear throughout these pages.

CONTENTS.

LECTURE I.

Extent and Physical Characteristics of British India—Climate— Rainfall—Effects of Irrigation—Population—Races—Habits— Food—Prevalence and Causes of Fever among different sec- tions of the Population—Etiology of Fevers—Nature of Malaria —The Bacillus Malariae—Modes and Conditions of Action of Malaria—Action of Malaria on the Lower Animals and on Man —Distribution of Malaria in India—Effects of Season.	PAGE 1
---	-----------

LECTURE II.

Peculiarities of Indian Fevers—Intermittent Fever as observed in India—Outbreak of Fever at Amritsar—Relative Frequency of the different Types—The Paroxysm described—Period of Incu- bation—Complications—Chronic Malarial Poisoning—Patholo- gical Anatomy—Malarial Pigmentation—Effects on the Spleen and Liver—Effects of Malarial Fever on Wounds—Cases of Intermittent Fever—Remittent Fever—General Description— Pernicious Forms—Masked Malarial Fevers—Malarial Cachexia —Treatment of Malarial Fevers—Treatment of Remittent Fever —Illustrative Cases.	58
--	----

LECTURE III.

Arrangement of Continued Fevers—Ephemeral Fever or Febricula —Ardent or Thermic Fever—Endemic Enteric Fever—Ques- tion of Specific Enteric Fever—Views of English, French, and American Observers—The Burdwan Fever—Etiology of Enteric Fever—Pathology—Symptomatology—Treatment—Illustrative Cases—Conclusion.	152
--	-----

INDEX	275
-----------------	-----

LIST OF TEMPERATURE CHARTS.

No.	1.	QUOTIDIAN	<i>Case</i>	II.	<i>Page</i>	88
	2.	QUOTIDIAN	"	III.	"	88
	3.	TERTIAN	"	VI.	"	90
	4.	QUARTAN	"	VII.	"	90
	5.	REMITTENT	"	XV.	"	122
	6.	REMITTENT	"	XVII.	"	126
	7.	CONTINUED REMITTENT	"	XIX.	"	128
	8.	REMITTENT	"	XXIII.	"	130
	9.	FEBRICULA	"	XLVIII.	"	234
	10.	FEBRICULA	"	XLIX.	"	234
	11.	SIMPLE CONTINUED	"	LI.	"	235
	12.	THERMIC FEVER	"	LII.	"	236
	13.	ARDENT OR THERMIC FEVER	"	LVII.	"	243
	14.	ENTERIC	"	LXI.	"	247
	15.	ENTERIC	"	LXIII.	"	251
	16.	ENTERIC	"	LXVII.	"	253
	17.	ENTERIC	"	LXIX.	"	254

THE CLIMATE AND FEVERS OF INDIA.

LECTURE I.

MR. PRESIDENT, Fellows of the College, and Gentlemen,—
In conformity with the custom of others who have been honoured with the duty of delivering these lectures, I have selected a subject in practical medicine on which to address you, that has been made somewhat familiar to me by experience in the East, and which will prove, I hope, of sufficient interest to merit your attention, not merely because it is the origin of much sickness and mortality among the European and Native people of India and is the cause, directly or indirectly, of most Indian diseases met with here; but on account of its relation generally to the cognate subject in Europe. I trust, therefore, that I shall give effect to the purpose of the founder of these lectures, and not altogether unprofitably occupy your attention, by selecting as my theme certain ‘Types and Forms of Indian Fever,’ premising a brief sketch of the physical characters of the country, climate, and people in and among whom they occur. My remarks will apply mainly to Indian fevers, but I shall refer to others when climatic or other conditions, by *determining a similarity of type, make it expedient to do so.* I venture to think that the time is not inopportune, for it is thought by some, of considerable experience, that the present nosological arrangement of fevers needs some

modification, as regards their etiological relations in India and the tropics, and that sufficient importance has hardly been attached to the effects of geographical position, climate, and endemic influences in determining and modifying their characters. To this subject, therefore, I propose to ask your attention, and the following is the plan I intend to adopt:—After giving a brief outline of the physical characters of the country, climate, and people, and of the prevalence of fever in British India, I propose, under the head of Malaria and Malarial Fevers, to describe the various remittent and intermittent forms they assume, from the deadly jungle fever of the primæval forests and swamps, to the simplest ague or malaise of the plains; and also some of those morbid conditions which, although they present little, if any, pyrexia, are so closely related, etiologically, to malarial fevers, that they are naturally considered with them.

Under Continued Fevers, I shall endeavour to include those not primarily malarial, such as typhus, relapsing, enteric, ardent, and other forms; comparing them with continued fever in temperate climates, noting their relations to climatic influences, and remarking that fevers, though varying in intensity, do not differ in essential characters from those of temperate climates—as may be deduced from the writings of men who, like Pringle, Fergusson, Lind, and others, studied them in former years, before sanitary science had attained its present development.

I do not pretend to have much that is new to say, for these diseases have been described by numerous writers, in a literature that is most copious and continuous from the earliest periods down to the present day. But in giving my own impressions, and in submitting any new views, it is expedient that I should refer to and acknowledge those of others from whom I have derived so much valuable information. The subject is so extensive that I can only hope to touch on the salient points of interest.

Let me remind you first of some of the physical charac-

ters of the country and climate in which these fevers occur. Health and disease are so greatly influenced by the locality, nature of the soil and its vegetation, the temperature and its fluctuations, the quantity and seasonal distribution of the rainfall, atmospheric and other meteorological changes, that some reference to them seems a fitting introduction to the study of the fevers themselves; for, though this is true of all disease, it is peculiarly so of fever. You will, I trust, pardon me for inflicting on you some geographical and statistical details, which I will make as brief as possible; they relate to a country and a people whose extent and magnitude are but too imperfectly appreciated, and have an important bearing on the subject to be submitted to you.

Physical Geography.

Our great dependency in Asia, British India, situated between the eighth and thirty-fourth parallels of north latitude, and the sixty-sixth and ninety-fifth meridians of east longitude, has a coast-line of more than 4,000 miles. It is 1,900 miles in length from Peshawur to Cape Comorin; and about the same in breadth from Assam to Kurra-
chee; it is 1,000 miles from Bombay to Calcutta. The superficial area is about 1,600,000 miles. The geographical boundaries are on the north the Himalayas, 150 miles in average breadth, extending north-west and south-east in a double crescentic range, and traversed by great rivers (Ganges, Sampu, Indus) which run east and west for 600 miles. Their mean height is from 16,000 to 20,000 feet above the sea level; Mount Everest, the loftiest peak, being over 29,000 feet high. This barrier, which separates India from Turkistan and Tibet, is crossed by passes 17,000 feet above the sea, nearly on a level with the line of eternal snow. On the north-west India is bounded by Afghanistan and Beloochistan, the Suliman and Hala mountain ranges; on the north-east by the heights of Assam, which divide the drainage of the Brahmaputra

from that of the Irawaddy. Its coasts have the Bay of Bengal on the east, the Arabian Sea and Indian Ocean on the west and south, enclosing a table-land of from 1,500 to 3,000 feet above the sea level, between the Eastern and Western Ghâts, which slopes gradually to the east, most of the rivers running eastward to the Bay of Bengal. The mountains are separated into two distinct systems by continuous lowland extending from the Arabian Sea to the Bay of Bengal. The north part of this lowland skirts the foot of the hills, and forms the pestilential malarious region called the Terai. The outer range of hills is known as the Siwalik, and Salt Range, and is about 2,000 feet high, with an intervening valley named the Dhoon. South of Hindostan is the table-land of the Deccan, extending through 20° of latitude. Its margins are the Eastern and Western Ghâts and the littorals of the Arabian Sea and Bay of Bengal. The Western Ghâts rise from 4,000 to 5,000 feet; Dodabetta, the southern peak in the Neilgherries, is 8,640 feet high. The Eastern Ghâts are not so high, and much less continuous than the Western. India forms two great watersheds; that of the Bay of Bengal on the east, that of the Arabian Sea on the west; the water-parting running nearly vertically from Cashmere to Cape Comorin. This vast country, with every kind of climate, possessing lofty mountains, elevated table-lands, alluvial valleys, desert tracts, and plains; noble rivers, estuaries, extensive swamps, jungles, and magnificent forests; has characters that invest it with peculiar interest for those who desire to study the influences of geographical position, geological formation, and productions of soil and climate, on the development of man, and the origin and diffusion of disease.

Climate.

As regards climate, India may be divided into three regions:—1. The Himalayan, which includes Bhotan, Nepal, Gurhwal, Cashmere, and Cabul. 2. Hindostan, which

extends along the foot of the Himalayan range, and includes the alluvial plains of the great rivers Ganges and Indus, with their numerous tributaries, as far south as the Vindyah mountains. 3. Tropical India, or the Decan, which consists of elevated table-lands, littoral plains intersected by numerous rivers, mountain ranges, extensive forests, and isolated hills.

There are three distinct seasons in India—the hot, the rainy, and the cold—which vary in duration and times of setting in; the cold season extends from November to March, the hot from March to June or July, and the rainy season from then to October or November; these seasons being greatly influenced by the monsoons or periodic seasonal winds. The south-west monsoon, which commences with storms of thunder and wind, soon followed by the bursting of the rain on the Malabar coast in May, does not reach regions farther north till later in the year; its force and influence, indeed, are well-nigh spent ere it passes the twenty-fifth parallel of north latitude. The Carnatic, and the Coromandel coast, sheltered by the Western Ghâts, are dry when the west coast is deluged with rain.

In the north-west the rains begin towards the end of June, and fall in diminished quantity. Near the hills the rainfall increases; but in the Southern Punjab and in the Great Desert there is very little rain—in some parts none. There are tracts of country, commencing in Sind, almost rainless, or with a fall as low as two inches; whilst in the Khasia hills, on the north-east frontier, 600 inches fall in the year. Next to this, the Western Ghâts and coast of Tenasserim have the greatest fall; at Mahableshwar 250 to 300 inches, and on the Tenasserim coast 180 inches fall yearly.

The amount of atmospheric humidity also varies greatly. Flat hot plains, like Sind, where there is little or no rain, have an atmosphere almost saturated with moisture, whilst on some of the lower mountain ranges, in Bengal, and in many districts near the coast in Southern India, the air is very damp. But on the elevated table-

lands of the Deccan and Central India, and the hot sandy plains of North-west India, a dry air blows like a furnace blast during the months of May and June.

The north-east monsoon commences in October, and is dry, except on the Coromandel coast, where it brings rain between October and December. Variable winds last till about June. About the end of May the south-west monsoon again sets in, bringing a few showers, known as the lesser rains, which precede the greater rains. In the hill stations of Darjeeling, Mussoorie, Nainee-Tal, Murree, Simla, and generally in the elevated provinces of the lower ranges of the Himalayas, also at Ootacamund, Conoor, Wellington, Mahableshwar, in the Neilgherries, and Ghâts—stations at elevations of 5,000 to 7,000 feet—the climate is genial, the rainfall moderate, it is cool and healthy in summer, and almost as bracing in winter as Europe. These may perhaps become the sites of future colonisation, for it seems probable that there the European may thrive and continue to reproduce his race, which, it is said, would cease to exist in the plains after the third generation.

The mean temperature of a few well-known stations is:—

Calcutta, 8 feet above sea level, in May (hottest month), 89°; in January, 70°; it ranges between 45° in the coldest and 92° in the hottest months.

Madras, sea level.—June (hottest), 88°; January, 76°. Range, 72° to 92°.

Bombay, sea level.—May (hottest), 86°; January, 74°. Range, moderate.

Peshawur, 1,056 feet above sea level.—June and July (hottest), 91°; January, 52°. Range, great.

Punjab, 900 feet above sea level.—June (hottest), 89°; January, 54°. Range, from frost to intense heat—110° and more.

Bangalore, 3,000 feet above sea level.—May (hottest), 81°; January, 69°. Range, moderate.

Poonah, 1,089 feet above sea level.—May (hottest), 85°; January, 70°.

Belgaum, 2,200 feet above sea level.—April (hottest), 81°; May 78°; June, 75°. December (coldest), 70°.

Rainfall.

A glance at a rain map of India shows areas of rainfall of various degrees, of irregular form and extent.

In the north-west corner of India there are arid regions, which have a fall of less than 15 inches ; in many parts much less ; whilst the desert tract of the Thar is to some extent rainless. This area includes Sind, part of the Punjab and of Rajputana. Then there is a zone with an annual fall of between 15 and 30 inches, surrounding the arid region on the north and east, in a belt of 100 to 200 miles wide, which includes Delhi and Agra. This is the northern dry zone. The upper parts of the valley of the Ganges, Central India, and the coast of the Madras Presidency, have a fall of between 30 and 60 inches.

There is also a southern dry region, which extends from Nassick to Cape Comorin, whilst the deltas of the Mahanuddi and Ganges, and the lower part of the Gangetic Valley, have a fall of between 60 and 75 inches. There are two belts of excessive rainfall, one extending along the Aracan coast, from the mouth of the Irawaddy up the valley of the Brahmapootra. The other, on the west coast of India, from Cape Comorin to the Tapti—from the seashore to the summit of the Ghâts. In these regions the most remarkable falls occur, for the reason that they are placed in the direct course of the south-west monsoon, and catch its first impact at heights where vapour is most readily condensed into rain. At Cherra Poonjee, in the Khasia hills, at 4,500 feet above the sea, 600 inches of rain fall in six months on the edge of an abrupt mountain ridge and plateau, situated about 200 miles from the Bay of Bengal, the intervening country being an alluvium, covered with rivers and swamps. Over this the south-west monsoon blows, laden with moisture, which is increased by the wet country over which it passes. At Mahableshwar, in the Western Ghâts, the conditions are somewhat similar, the fall amounting to about

300 inches; but these excessive rainfalls in certain elevated regions are quite local, no more representing the average rainfall of all India than does the dryness of the desert tracts in the north-west of that country or the heavy fall on the hills on the west coast of Britain, in Cumberland, or Scotland, represent the average rainfall of Great Britain.

The following are averages of a number of previous years:—

In Calcutta, the whole rainfall was 65·80 inches, the greatest falls being in June, 11·78; July, 12·77; August, 13·96; September, 10·15.

In Chittagong, the greatest falls were—in June, 21·35; July, 21·93; August, 21·71; September, 14·05. The whole rainfall was 103·7.

In Bombay, the whole rainfall was 74·20. The greatest was—in June, 20·95; July, 24·27; August, 15·21; September, 10·71.

In Kurrachee the fall was 7·61. The greatest being—in July, 2·97; August, 2·10; September, 0·81; December, 0·22; January, 0·67; February, 0·26.

In Mangalore, on the west coast, in the full intensity of the south-west monsoon, the fall was 134 inches. The greatest falls were—in June, 40·09; July, 37·68; August, 23·14; September, 11·70; October, 8·55.

In Madras, 48·15 fell. The greatest falls were—in October, 10·73; November, 13·0; December, 4·99; January, 0·65; showing the influence of the wet north-east monsoon.

In Tinnevely the fall was 28·16 in the whole year: greatest—in October, 6·25; November, 9·86; December, 2·63; January, 1·55.

In Southern India, at several stations—as, for example, Coimbatore, Bangalore, and others—both monsoons are felt, and a certain amount of rain is due to each.

Irrigation.

Though great part of the continent of India is amply supplied with rain, there are extensive regions where the normal quantity is so small that it is insufficient to produce the crops necessary for the support of the population,

and where, without the aid of artificial irrigation, the land would be sterile. This is effected by reservoirs, canals, and wells.

The Government of India has given much attention to artificial irrigation, and many gigantic works have been completed, whilst others are in course of construction for this purpose; some are new, others reconstructions on former lines of works of the Hindoo and Mahomedan periods; and the importance attached to irrigation is manifested in the canals, anicuts or dams of rivers and reservoirs, many in ruins, left by these people.

There are 12,750 miles of lesser or greater canals, whilst the total length of the distributing canals is unknown. In Northern India alone, however, it amounts to 8,300 miles. The area now irrigated amounts to 1,900,000 acres in Madras and Bombay, 300,000 in Behar and Orissa, 1,450,000 in North-West Provinces, 1,350,000 in Punjab, and 1,250,000 in Scinde; in all, 6,310,000, nearly six and a half millions of acres. The area irrigable by canals is yet considerably greater than even this large total, so that the system is capable of extension. The agricultural benefit derived from this system of irrigation is no doubt great, but it has disadvantages, for with the water there is generation of malaria and fever, to what extent I shall presently show.

Another point to which I would refer very briefly is the influence of the rainfall on the growth of forests, and the effects of these on climate. There is reason to believe that some of the desert plains of India were at one time covered with trees, and that when they were so the climate was less excessive in its extreme heat than it is now. The desert regions in the north-west having been the seat of early Hindoo civilisation and population, it is obvious that the physical conditions of the country must have once been very different to what they are now, and it seems probable that the change is in some measure due to the destruction of trees. The cultivation and protection of existing forests, therefore, is a matter of the

greatest importance, for, not only do they temper the climate by the moisture they exhale, but they tend to cause rain where there would be none.

Population.

The population of India, according to the last census, of February 1881, is 252,541,210; the males appear to exceed the females by about 1-50th. Notwithstanding all checks, it has increased about $12\frac{1}{4}$ millions since the census of 1872, at the rate of $6\frac{1}{2}$ per cent., except in British territory in Bombay, in Mysore and Madras, where there is a decrease of from 20 to 2 per cent., owing to famine and its consequences. The census of 1872 returned roughly 239,750,000 inhabitants, viz. :

Hindoos and persons of Hindoo origin	149,130,185
Mahomedans and persons of Mahomedan origin	40,227,552
Asiatics (not natives of India)	540,989
Of mixed races	108,402
British	75,734
Other Europeans	8,000
Europeans (unspecified)	30,453
Americans, Africans, Australians	6,961
Other unspecified non-Asiatics	434,772

To which must be added the residents in feudatory States.

The average of the whole population gave about 162 per square mile; but in the British territory it was twice as dense as that of the feudatory States, the number of persons per square mile varying considerably in different provinces.¹

This vast mass of human beings, double the number of the population of the Roman Empire in the zenith of its power, and larger than the population of all Europe (excluding that of Russia), is composed of many races differing among each other, more than do those of

¹ In Bengal it was 387; in Bombay, 131; in Baroda, 454 per square mile; whereas some native States in the Central Provinces had only 36 to the same area. Many causes, no doubt, combine to explain these differences.

Europe. They inhabit a continent presenting every variety of physical geography and climate, and almost every physical condition on which health may depend, or by which disease may be originated and propagated.

The people of India represent four great classes :—

1. The non-Aryan, composed of Turanian and Dravidian or Scythic stock, the aborigines so-called, and their descendants ; amounting roughly to about eighteen millions.

2. The Aryan, represented by the high-caste Hindoos, Bramins, and Rajpoots ; about sixteen millions.

3. The great mixed population, commonly known as Hindoos, grown out of admixture of Aryan and non-Aryan elements ; about one hundred and ten millions.

4. The Mahomedan Semitic races, who invaded India from the north and north-west ; about forty-one millions.

To these must be added the Eurasians, 108,402, and the European races who now rule India, 121,148. I cannot stay to trace the early history of these races ; but, as regards their distribution, may just say that the Himalayas are occupied chiefly by the descendants of the Turanian stock, the plains of Hindustan by the Aryan race, and the table-land of the Deccan by a mixture of Aryan and Scythic or Dravidian races. The Europeans and Eurasians are distributed over the whole continent.

Among this diversity of races there is equal diversity in physical characters. A Norwegian does not differ more widely from a Neapolitan than a Sikh or Pathan from a Bengali, or the powerful warlike Rajpoot of the North from the peasant of Southern India. In mental and physical attributes there is a close analogy ; and, corresponding with the difference of climate, locality, and habits, they evince different capacities for enduring or resisting disease ; some, as I shall show, having a peculiar power of resisting malaria.

Habits and Food.

With regard to their habits and food : the Mahomedans, Sikhs, some of the lower castes of Hindoos, and

the aboriginal races, eat a mixed diet, including animal food, and are often robust, powerful people; many are in military service. The higher caste Hindoos subsist mainly on farinaceous food, such as wheat, rice, millet, and other similar grains, supplemented by pulse (dhal, a sort of pea), with milk, ghee, vegetables, and fish—occasionally the flesh of the goat or pigeon. Those who inhabit northern India are powerful men, and from them the army is largely recruited.

Among the Europeans and Eurasians there is, I think, a tendency to eat more animal food and to drink more alcohol than are absolutely required, though in both respects they are more judicious than in past years. The natives of all classes are, as a general rule, temperate as regards stimulants; though some races, such as the Sikhs, the low-caste Hindoos, and the aboriginal races, drink freely. The use of tobacco, hemp, betel, and even opium—the latter especially in malarial districts—is very prevalent; opium, however, is much less used than among the Chinese.

On the whole, the natives of India are moderate and abstemious as regards food and stimulants—rather under- than overfed, especially among the lower classes; and therefore more prone than the European to the asthenic condition in disease. As to clothing and habitation, these are of the simplest kind; the dress for the most part is of cotton, which in northern regions is supplemented by warmer materials. The dwelling of the ordinary native is generally a hut; among a large proportion of the people, built so near the ground and so ill-ventilated that miasmata play a great part as a cause of disease. The lodging of the European is, as a general rule, good; sanitary precautions are duly observed, and the houses, barracks, clothing, food, and occupations of the British resident and soldier are so well-ordered and carefully supervised as to leave little to desire. When this is taken into consideration, together with the fact

that the soldiers are young, selected, and healthy, their diseases may be regarded as a test of the real operation of malaria uncomplicated by other causes.

The habits and social conditions of a large proportion of the native people are prejudicial; early marriages, and sexual excesses and abuse, play an important part in sapping the vital force. The effects of impoverished health and vitality, and of the diseases that result from imperfect nutrition, have been painfully manifested in the results of the famines that of late years have ravaged large districts and checked population, whilst the great prevalence of malaria is shown in the character of the fevers and bowel complaints which destroy or deteriorate millions, and cause the native to suffer far more than the European.

It is satisfactory to know that under the fostering care of the British Government gradual amelioration is progressing, and that already among the natives, as well as the Europeans, the intensity and mortality of disease is diminishing, and the mode of living is being improved.

Prevalence of Fever, and its Causes.

Let me now speak of the extent to which fever prevails, and some of the reasons why it does so. Official records afford proof that it causes an amount of sickness and mortality which is hardly credible, and in some years almost challenges comparison with the Black Death which ravaged Europe in the fourteenth century and destroyed a fourth part of the whole population. The registered deaths from all causes in India in the year 1879 were 4,975,042:

Cholera accounting for	270,552
Small-pox accounting for	194,708
Bowel complaints accounting for	250,173
Fevers accounting for (out of a population of 187,105,833)	3,564,035

Civil Population, Fever Deaths.

1877	2,504,493
1878	3,247,371
1879	3,564,035

or thirteen times as many as from cholera; though it may probably fairly be estimated that not more than 50 per cent. of these deaths were due to endemic fevers.¹ In the case of certain classes subject to registration, the figures are reliable; those relating to the general population are probably less trustworthy, but still sufficiently accurate to give a tolerably correct idea of the prevalence of disease and the extent of mortality.

Let us look at the statistics of fever prevalence as illustrated by the sanitary reports and returns of hospitals in Calcutta.

The mortality from 'fevers' in Calcutta has been during six years as follows:—

1875	.	.	5,328	1878	.	.	6,186
1876	.	.	4,361	1879	.	.	4,796
1877	.	.	5,151	1880	.	.	3,797

Clarke says that in 1770 80,000 natives and 1,500 Europeans died from fever in the city of Calcutta.

¹ In the present state of registration it is not possible to define the special character and type of these fevers. They are certainly, for the most part, malarial in character. Attempts have often been made to arrive at the actual number of true fever-deaths, and they all agree in showing that fatal diseases attended by heat of skin and other febrile characters are returned as fevers. In the Chanda district, out of 1,008 deaths, the Civil Surgeon found that 672, or 66·6 per 100, were due to fevers properly so called. The Civil Surgeon of Betul reports that out of 208 deaths, 66, or 31 per cent., were fever deaths; and so on. The Civil Surgeon of Mandla states that from personal inspection he has ascertained that fever forms 7·5 per cent. of the mortality. The Civil Surgeon of Wardah gives the following result of personal inquiry into the cause of 69 reported fever deaths.

18 died of Remittent	2 died of Pleurisy
9 died of Bronchitis	2 died of Peritonitis
8 died of Ague	3 died of Vermes
3 died of Continued Fever	

Suppose 50 per cent. die of fever, the loss of life is great.

The death-rate from fevers at the Medical College Hospital has been:—

	Christians	Natives	Total
1878 .	35.2 .	201.9 .	237.8
1879 .	46.05 .	121.09 .	167.14
1880 .	37.7 .	89.0 .	126.7

The total number of cases treated in the Medical College Hospital (including in and out patients, Christians and natives) of intermittent, remittent, continued, and febricula during the past ten years was:—

1871	7,869	1876	7,582
1872	7,600	1877	8,023
1873	7,595	1878	9,229
1874	8,492	1879	7,936
1875	7,438	1880	7,191

Statement showing the number of cases of remittent fever and typhoid or enteric fever treated in the principal Calcutta hospitals in the preceding ten years:—

REMITTENT FEVER.

	1871	1872	1873	1874	1875	1876	1877	1878	1879	1880
Medical College .	42	80	86	58	125	95	124	152	164	180
Presidency General Hospital .	45	19	22	10	48	27	39	38	34	49
Howrah Hospital .	16	28	88	146	172	210	359	393	228	107
Mayo Hospital .	6	15	13	22	35	36	174	224	130	208
Campbell Hospital .	181	239	212	495	466	438	432	466	333	354
Police Hospital .	—	—	—	—	1	—	—	11	29	28
	290	381	421	731	847	806	1128	1284	918	926

TYPHOID OR ENTERIC FEVER.

	1871	1872	1873	1874	1875	1876	1877	1878	1879	1880
Medical College .	3	5	9	5	6	6	1	—	2	—
Presidency General Hospital .	15	17	6	11	12	17	18	9	11	6
Howrah Hospital .	—	—	—	—	—	—	4	2	4	2
Mayo Hospital .	—	6	2	1	5	6	—	—	—	—
Campbell Hospital .	94	86	28	2	—	—	—	—	—	—
Police Hospital .	4	12	8	11	—	14	11	2	1	—
	116	126	53	30	23	43	34	13	18	8

Professor McConnell, who has obtained the statistics for me, remarks :—

‘A comparison of these tables is very interesting, as showing the relative frequency of typhoid and remittent fever in various years, those being the two kinds of fever often so difficult during life to differentiate. The larger preponderance of typhoid in the earlier years at the Campbell Hospital is due to the inclusion of many cases of remittent fever; and the same may be said of the statistics of the General Hospital, where, owing to the better class of patients treated (almost all Christians and no natives), *post-mortem* examinations are frequently not permitted by the friends, and thus the verification of the diagnoses during life is not obtainable.’

The British army in India in 1879 numbered 57,810 men, and of these 51,959 suffered from fever, with a mortality of 387.¹ The native army, of 130,011 men, had 122,375 cases of fever with 756 deaths. The jail population, of 117,680 persons, had 73,484 cases of fever with 1,306 deaths. 1879 was an unusually unhealthy year, when epidemic fevers of a malarial type were prevalent and fatal. In some districts, during and after the close of the rains and beginning of the cold season, the mortality was very high. In Bolundshur and Allyghur the

¹ The mortality of the British troops in the Bengal army during sixteen years, 1830 to 1845, gave a ratio of 13·25 per 1,000 from all fevers.

The same for 1875 was only 2·77 per 1000.

“	“	1876	“	2·46	“
“	“	1877	“	2·21	“

In 1878–79 there was more fever from the famine and Afghan War. These three years appear to be normal.

Army of all India for five years, 1871–75 :—

Deaths from all fevers, 1875 = 2·81 per 1000.

“	“	“	1876 = 2·41	“
“	“	“	1877 = 2·16	“
“	“	“	1878 = 5·07	“
“	“	“	1879 = 6·55	“

The rates for 1878–79 were part of the great fever epidemic which swept over India and occasioned enormous losses to the civil population. The rates include the Afghan fever and those during the march of troops.

deaths rose to about 113 per 1,000 of population, the mortality from all causes being but little in excess of that of fever alone.

The population of Bengal, under registration, in 1880 was 59,890,237; about 4 per cent. located in towns, 96 per cent. in country and villages. The deaths registered in the whole province were:—

Cholera . . .	39,643
Small-pox . . .	22,953
Fevers . . .	689,605
Bowel complaints	44,969
Injuries . . .	22,339
All other causes .	103,124
<hr/>	
Total registered deaths .	922,633

Fevers destroyed nearly three times as many as all other death causes put together!

THE PROPORTION OF DEATHS REGISTERED IN TOWN AND COUNTRY.
RATIO PER 1,000 POPULATION.

Diseases	Town	Country
Cholera	1·37	0·63
Small-pox. . . .	0·45	0·38
Fevers	15·16	11·36
Bowel complaints . . .	3·53	0·60
Injuries	0·47	0·36
All other causes . .	5·87	1·55
<hr/>		
All causes . . .	26·85	14·91

In the Bombay Presidency in 1880 the annual mortality from fevers averaged 193,508 during a period of fourteen years; but in 1880 the deaths were 246,779 or 15·21 per 1,000 of the population; *i.e.* for every fatal case of cholera there were 360 deaths from fever.

BOMBAY.

Seasons	Registered Fever Deaths	
	1880	Mean of 14 years
Cold.	93,800	63,446
Hot and dry	38,488	28,304
Rainy	73,746	62,992
Hot and damp	40,745	38,773
Total	246,779	193,515

The Deaths for each month being—

January	24,689	July	18,922
February	22,625	August	19,856
March	23,921	September	18,396
April	19,901	October	18,406
May	18,579	November	22,339
June	16,572	December	22,565

The seasonal prevalence (*vide* table) shows how cold and variable temperatures affect the etiology of these fevers. The contrasted mortality between that of the hot and dry, and the rainy and damp seasons, exhibits the effects of added moisture on the amount of fatality. The fevers in these returns are all placed under one general heading—the types comprehended being the malarial and continued. Among them, no doubt, are included deaths due to other inflammatory disorders, such as pneumonia; whilst on the other hand not a few of the deaths from dysentery and diarrhoea belong more properly to malarial fevers. Registration is rapidly improving, but is not yet perfect.

The thirteenth annual report of the Sanitary Commissioner of the North-West Provinces for the year ending December 31, 1880, contains much information in respect of the prevalence of fever, the causes of malaria, and the influence of irrigation on fevers. These provinces include

the pestilential Terai, the Doab, and the irrigated area, which was scarcely less unhealthy.¹

Population of Oude and North-West Provinces, 44,107,061 in census of 1881. The registered deaths for five years, including the period of scarcity, were :—

Years	Total Deaths	Deaths per 1,000 population
1876	937,490	21·94
1877	840,538	19·67
1878	1,521,724	35·65
1879	1,914,499	44·81
1880	1,281,155	29·99

The improvement in 1880 follows relief from scarcity. The lowest death-rate in 1880 was 21·72 in Rai Bareilly. The highest in the Terai 53·41.

The chief death-causes in 1880 :—

Diseases	Total Deaths	Deaths per 1,000
Cholera	71,546	1·67
Small-pox	8,240	0·19
Fevers	987,220	23·11
Bowel complaints	80,312	1·88
Injuries	20,553	0·48
All other causes	113,284	2·65

About twenty-three out of every twenty-nine deaths were due to fever. The mortality caused by small-pox, cholera, and dysentery, was nothing in comparison with that from fever! Nearly a million of people (987,220) died in 1880 of malarial diseases. The liability to fever

¹ 'The unusual rise in the fever mortality was marked in August and September, was at its height in October and November, and then gradually declined' (Sanitary Commissioner's Report for 1879, page 78). The Sanitary Commissioner of the North-West Provinces remarks: 'Everywhere the rise, culmination, and decline happen at the same time, and as from a cause co-existent everywhere in the Province. This cause was malaria, and it is attributed to excessive rainfall following two years of comparative drought' (Sanitary Commissioner's Report in 1879, Page 78).

here seems to have been increased by other predisposing causes, the most important being underfeeding; but as this was not the case with the entire population, and as they were not all badly clothed and housed, though all suffered, it is evident that there were other causes in operation.

The Sanitary Commissioners' Report for 1879 shows that the general causes which influenced the public health in fever localities were undrained ground into which canal water had been led, and rainfall added to the already water-logged subsoil. In 1880 food became cheaper, and except in certain districts there was less rain. In 1879, during the great scarcity of food, the fever death-rate was 37·82 per 1,000; in 1880, it fell with increase of food to 23·11 per 1,000, which was still above the five years' average of 20·91 per 1,000.

The monthly fever mortality for the whole registration area was:—

North-West Provinces, 1880.

Months	Fever Deaths	Months	Fever Deaths
January . .	116,366	July . . .	56,502
February . .	72,030	August . .	74,127
March . . .	69,250	September .	87,618
April . . .	72,534	October . .	91,248
May	76,622	November .	99,459
June	78,200	December .	93,264
Total . . .	485,002	Total . . .	502,218

The total annual deaths from fever taken through the scarcity period were:—

1877	574,722	1879	1,616,108
1878	982,117	1880	987,220

The fundamental cause of the great loss of life from fever was increased predisposition from scarcity of food; but cold, damp, and alterations of temperature in the latter half of the year, and dampness of soil, the result of irriga-

tion throwing more water into the subsoil than was needed by growing crops, must also have acted as serious predisposing causes.

The Chief Engineer was of opinion that the fever was *not* due to irrigation, but to great diurnal range of temperature, chills, and imperfect feeding. He says: 'Without the great diurnal range, canal irrigation will not produce malarial fever;' but he admits that under these climatic influences it may do so, especially in the winter months, and thinks that better clothing and food would protect the people. But we know that fever occurs irrespective of changes of temperature, though, no doubt, they have much influence in re-exciting it in those who have previously suffered, although they will not produce it *de novo*. Fever occurs in every month of the year, though more in some seasons than others. With the excessive mortality in irrigation districts, the conclusion is inevitable that the true cause is stagnant subsoil water. The remedy for all this is better drainage, whilst no more water should be used than is required for the crops.

The general unsanitary conditions which prevail everywhere at present, and were well described so long ago as 1768 by Sir J. Pringle (in the second chapter of his 'Diseases of the Army'), but which we hope gradually to ameliorate by sanitary work and by a more enlightened intelligence on the part of the people, are also among the predisposing causes that influence the severity of fevers.

Such is the state of prevalence of the diseases of which I propose to speak. The subject is of great interest, and is worthy of study by the physician who has to treat them, or the sanitary officer who brings all the resources of science to his aid in preventing or mitigating them.

With this brief introduction I pass on to consider the proper subject of this lecture, the fevers themselves, but first I must treat of malaria.

ETIOLOGY OF FEVERS.

Theories of the Origin of Malaria—Nature and General Characters of the Country.

India represents almost every variety and condition of soil and climate. In the southern and tropical parts, in the forest and jungle districts, on the littorals, and on the deltas of the great rivers, with an alluvial or dark loamy soil, heat and moisture generally prevail; on the elevated tracts and great desert arid table-lands, with a rocky or sandy surface, there is a dryer though still heated air; whilst on the hills, at various elevations, the peculiarities appertaining to the rarified, cooled, and tempered air of mountain ranges are found.

The influence of the monsoons is most marked within the tropic, and the effects of these great producers and distributors of rain have been referred to. The temperature of Southern India is comparatively equable, though high, the isothermal lines being influenced by elevation; and, except on the higher table-lands and on the hill stations, the general character of the climate, vegetation, people, and their surroundings are tropical, whilst the endemic diseases are, to a great extent, of a tropical or malarial type.

North of the Nerbudda and Vindyan hills, on the lower lands of Hindustan, along the basin of the rivers of the Punjab, the Indus, Ganges, and Brahmapootra, and on the extensive alluvial plains gradually accumulated by these great rivers, we have many phases of climate, and of extreme diurnal ranges of temperature. Bengal is hot and damp, with a prolonged rainy season and a delightfully cool cold season. North-west India, Oude, the North-West Provinces, Central India, and the Punjab, with much rocky and sandy territory, have a climate so cold in winter that frost is frequently seen, but a hot season of great intensity, with dry burning winds, great alternations of temperature or varying states of atmospheric humidity,

and a comparatively brief and scanty supply of rain. The hill stations of the Himalayas and other mountain ranges, at elevations of 5,000 to 7,000 feet, are cool and bracing, and are the coveted abode and health resorts of Europeans in India. The differences in climate are thus very great, and are especially noticeable in the dry heated air of Northern India, as compared with the steamy atmosphere of Bengal and the southern districts, the former being far more tolerable than the latter. They influence not only the physical and moral attributes of the people, but modify the character of their diseases, and especially that of the endemic fevers.

Much might be said on the relative effects of solar heat and light, moisture or dryness, and electrical states of the air, amount of ozone, the various soils, excess of vegetation or its absence, atmospheric pressure, vapour-tension, and the like; but I must pass on to consider the more special causes of disease which, in our ignorance of their real nature, we call malaria. Everywhere this exists, but in certain regions more intensely than in others. The low-lying jungle land, where the subsoil moisture is near the surface, at the foot of the Himalayas, known as the Terai, abounds in it, as is the case with the alluvial basins, silted-up beds and debouchures of rivers, water-logged soil of land in which watercourses have been obstructed, littoral plains, and in some regions sandy deserts. There are regions, that have formerly been populous, now deserted, and given up to wild beasts and malaria, which probably desolated them, such as the ancient city of Goa, and other cities of which the ruins are hardly to be traced in dense jungle.

Let me here observe that much is often attributed to climate which is more properly chargeable to defective hygiene and careless mode of living. Malaria prevails almost universally in India, at all events periodic fevers occur everywhere, and there are extreme vicissitudes of temperature and endemic causes of dangerous disease. But with due care, temperate living and ordinary precaution as

regards exposure to heat and to obvious causes of disease, the climate itself is less noxious than might be supposed. Let it be remembered that sanitary work, by removing these preventible causes, has already reduced the death-rate of European soldiers in India from sixty to sixteen per 1,000, and that it is proving gradually as beneficial to the civil population.

Nature of Malaria.

MacCulloch says: 'Perhaps the best, as the truest, account of the nature of malaria would be an acknowledgment of utter ignorance,'¹ and I fear we have not much to add to this description of a hypothetical cause of disease which is of very general prevalence, according to Lombard, between the 65th parallel of north and the 25th or 30th of south latitude, and which has baffled and puzzled mankind as to its nature since the earliest days of medical science. But it is well known by its effects on human health and life, these effects being manifested in all climates whenever certain conditions favourable to their development are called into existence, and varying in intensity from the deadliest fever to the most transient disturbance of the general health. It has been estimated to cause half the mortality of the human race, and has been called 'the great enemy, the very Destroying Angel, to whom the task of keeping man within bounds has been specially assigned.'²

In tropical climates it attains its greatest intensity, is the cause of several forms of fever, and seems to be intimately connected with the etiology of cholera, dysentery, hepatic disease, bowel complaints, and other morbid conditions; but in temperate, even cold climates, under certain conditions, its effects are also manifested. It abounds in Southern Europe, and our own islands are not exempt; though the agues that prevailed in London in Sydenham's time are now—thanks to sanitary science—matters of

¹ *On Malaria*, p. 419.

² *Ibid.* p. 458.

history ; but its influence still lingers in certain districts, in the eastern or southern counties, such as the fens of Lincolnshire and the marshes of Essex or Kent. The writings of Pringle, Fergusson, Blane, MacCulloch, and many others, describe its effects in Europe as having been formerly of great severity.

With the view of showing the decline of malarial fever in this country I have consulted the records of one of our great metropolitan hospitals ; and I am indebted to Dr. Stephen Mackenzie for a return which must have cost much labour. The London Hospital, from its size and position, is well qualified to afford information on this subject, and its records show the diminution that has taken place during the past century in malarial disease in the vicinity of London, proving that subsoil, sewer and surface drainage, better living, with the other sanitary improvements that have been in progress, have nearly extinguished malaria. The return shows the mortality, prevalence, and type of fever, and that the quantity, as well as the intensity, have diminished. The same process, we hope, is going on in India, and there is reason to believe that as sanitary knowledge becomes more a part of popular education and belief, further decrease of sickness and mortality will be the result.

The table (which was exhibited at the lecture) shows a considerable decrease in the number of fever admissions. In 1770, of 1,483 patients admitted, there were 21 cases of intermittent, with one death. 1780 seems to have been an unusually unhealthy year. There were, among 1,617 persons, 59 cases of intermittent fever, but only one death. 1779, with 1,886 in-patients, had 40 cases of intermittent, with four deaths. 1790, with 1,585 patients, had four cases of intermittent. (The register is imperfect up to 1830.) In 1840, with 3,337 in-patients, there were six cases of intermittent, no deaths. In 1870, with 5,218 in-patients, there were only two cases of intermittent. In 1880, with 6,312 in-patients, there were 14 cases but no deaths.

Before Hippocrates wrote on epidemics it was known that people who lived in marshy districts were liable to

suffer from intermittent fever. But it was not until the end of the seventeenth century (1695) that Lancisi, in Rome, wrote his great work on marsh exhalations, in which he pointed out their connection with these fevers, and called them paludal or marsh miasmata. The term is still in use, though the modern word 'malaria' is more generally preferred—that of paludal being apt to mislead, by indicating marsh miasmata as the only cause, whereas paroxysmal fevers are often met with on a dry, sandy, and rocky soil, where no marsh exists. Still, experience teaches that for their production the presence of moisture, organic matter, and a temperature above 60°, are generally requisite. Whatever be the cause, it is abundant in India, both in marshy and jungly and in arid and elevated regions. Until quite a recent period it was held to be of an aeriform or gaseous nature; though, since the time of Varro (114 B.C.), Lucretius (95 B.C.), and Columella (50 A.D.), Kircher (1602) and Linnæus (1778), it had been surmised that some low form of organism might be concerned in its production. In 1866, Dr. Salisbury, an American physician, thought he had discovered it in a palmella associated with cells and sporules of other fungi, though the latter were not constant, to which he gave the name of gemiasma or ague plants. Mitchell, of Philadelphia, had previously, in 1859, endeavoured to show a cryptogamic origin of malaria.¹ Balustra discovered a species of alga in the Pontine marshes.² Niemeyer also ascribed malaria to low organisms. Harkness found the palmella and spores in snow on the summit of the highest Alps, and says that they may readily become mixed with saliva and urine from without, and have nothing to do with malaria.³

When investigating the causes of intermittent fever in the valleys of the Ohio and Mississippi, Salisbury detected algæ in the mouth and air-passages of per-

¹ *American Journal of Science*, January 1866.

² *Comptes Rendus*, tome lxxi. p. 235.

³ *Boston Medical and Surgical Journal*, January 14, 1869.

sons suffering from ague. He found that individuals who slept in a room in which earth containing these sporules was placed, contracted fever, though they were five miles distant from any other known source of malaria; he repeated the experiment with similar results. This view of malaria seems soon to have been consigned to oblivion, though a vague impression remained that organic germs might have something to do with it.

The subject has recently been revived by Klebs, Tommasi-Crudelli, and others. In the year 1879 they announced the discovery, in the soil, water, and air of the Roman Campagna and marshes, of germs or sporules which are capable, under the influence of a temperature above 20° Cent., moisture, and air, of rapidly developing into sporigerous bacilli (the absence of any one of these conditions is fatal to their development); and it is stated¹ that the bacilli or spores have been found in the marrow of bones, the spleen, and blood of persons dying of pernicious fever. These bacilli they consider to be malaria, and the results of their investigations were summarised in the 'Medical Record' as follows:—

1. The poison is contained in great quantities in the soil of malarial districts at seasons when no fever is prevalent.

2. At such times it can be collected from the *air* immediately above the soil by an aspirator.

3. Stagnant water does not appear usually to contain organisms peculiar to malaria.

(a) Rabbits inoculated with washings of soil, or with fluids in which the bacillus had been cultivated, suffered from intermittent fever, the interval being in some cases 80 hours.

(b) *Filtered* liquids caused only slight symptoms, even if five times the original quantity was used.

(c) All the animals with intermittent fever had marked splenic enlargement, nine or ten times the normal size.

(d) Many of the spleens contained black pigment, especially those from graver cases, just like spleens of persons suffering from ague.

¹ *Medical Times and Gazette*, January 17, 1880.

- (e) The bacilli were found in the spleen and marrow of animals, as well as in the soil. They were at first ovoid, mobile, shining spores, which developed in the body, as well as in cultivation apparatuses, into long threads, homogeneous at first, but soon dividing into sections, each of which give rise to a new thread.
- (f) These bacilli could not develop without oxygen, and required a richly nitrogenised medium for their growth and cultivation.

Tommasi-Crudelli, in a note published in the 'Gazzetta Medica Italiana,' October 1880, summarises the observations made under his directions by Perroneito, Marchiafava, Cuboni, Ceci, and the experiments of Valenti, Ferraresi, Sciamanna, and Piccirilli.

Their results are:—

1. The bacillus may be found in any quantity in the soil of the Campagna or Pontine Marshes, and may be grown freely in that medium; it could not be got from soil taken from healthy places in Lombardy.

2. During the hot weather the air in malarial districts was so charged that numerous bacilli could be found condensed in the sweat on the hands and face of persons working there.

3. Spores of the bacillus were constantly present in the blood of infected rabbits, in that of persons suffering from ague, and in blood aspirated from the ague patients' spleens during the acme of fever. These spores when cultivated gave fully-developed bacilli, while those from other patients gave negative results.

4. Venous blood from ague patients gave intermittent fever to dogs when subcutaneously injected.

5. Blood removed during *invasion* always contained numerous bacilli, but at the acme they disappeared, and only spores could be found (similar to the behaviour of spirillum in relapsing fever).

Tommasi-Crudelli thinks that the rigors may be produced by the irritation of the vaso-motor nerves by the bacilli in the circulation, that the febrile attack is the result of the discharge of the bacilli from their special *nidus* in great quantities, and that their development and disintegration are accelerated by the high temperature, abundant nourishment, and oxygen of the blood.

Lanzi, Terrigi, Marchiafava, Cuboni, and some French

observers in Algeria, have confirmed these observations. They say they have been able to communicate true malarial fever to dogs and rabbits, by inoculating them with blood taken from malaria patients. They have found the bacillus in the blood of malarial cases, sporadic cases also, a constant fact. It is found in great quantities in the blood during the cold stage, but it disappears in the hot stage, almost totally, leaving only traces of its spores, which in their turn produce a second generation of parasites. Lanzi, of Rome, says he observed the same fact in the blood of twelve patients in the Hospital of S. Giovanni in Laterano. Following on this, Professor Tommasi-Crudelli has suggested, that arsenic should be used as a prophylactic to render the human organism insensible to malaria.

Laveran¹ has found organisms in the blood of malarial subjects—cylindrical curved bodies, pointed, transparent, but with a pigmented spot, without movement; also cylindrical bodies, about the diameter of a red corpuscle, containing pigment granules, presenting rapid movements. On the borders of the spherule were filaments in rapid movement, three or four times the length of the diameter of the corpuscles. There were also bodies of spherical or irregular form, transparent or finely granular, about the 100th of a micro-millimètre in diameter, containing dark-red, rounded pigment grains, they were motionless, and appear to be the ultimate stage of the above, have no nuclei, and do not tint with carmine-like pigmented leucocytes. Spherical elements, similar to the last, but smaller in size, were also present. The animated nature of the mobile pigmented spherules with filaments appears indisputable.

Laveran regards them as a form of animalcule, which exists at first in an encysted state, and in the perfect condition becomes free in the form of mobile filaments. Further, the blood of persons suffering from malarial fever contains (1) red corpuscles, which appear to be vacuolated

¹ *Lancet*, Nov. 12, 1881.

at one or two spots, and contain pigment granules; (2) pigmented leucocytes; (3) free pigment granules, probably proceeding from the destruction of the parasitical organisms. He discovered them about fifteen months ago, but has since found them in 180 of 192 persons affected with various symptoms of malarial poison in Algeria and Tunis. He is convinced that they are not found in the blood of persons suffering from diseases not of malarial origin. In the malarial cases where they were not found, it was when quinine had been previously taken. The addition of a small quantity of quinine to the blood destroyed the organisms. He thinks that during a pyrexial period the organisms probably sojourn in the internal organs, especially in the spleen and liver. After death from malarial disease, pigment granules are found in great numbers in the blood, and especially in the small vessels of the liver and spleen; even the marrow of bone and the brain substance are discoloured by their presence. These pigment granules, which may obstruct the capillary vessels, appear to be derived from the parasitic elements, which perish after death, and become unrecognisable. This also, I think, we must accept with reserve, but there is no doubt that micro-pathology is making wonderful advances, and that it is opening up new possibilities daily as to the origin of disease. In reference to the relation of minute organisms to the etiology of certain forms of disease, I remarked years ago that 'when infection has occurred, we need some effective parasiticide. It may be that some of the antiperiodic remedies—quinine, arsenic—have owed some part of their virtues to anti-hæmatozal properties.' I was referring to the *filaria sanguinis hominis* of Lewis and Manson, but if Laveran's discoveries be confirmed, the remarks would apply to them also. We seem to be on the threshold of the discovery of unknown and almost unsuspected disease-causes, and must watch the progress of investigation with great interest. One cannot help wondering where Tommasi-Crudelli's bacilli were in Laveran's cases, for he does not appear to

have seen them. I think we can hardly avoid a little scientific scepticism in these matters as yet.

Kelsch and Kiemer found that in the liver and blood of Algerian patients dying of malarial cachexia, the red corpuscles became larger and their number diminished. In a case of quotidian ague there was a loss in twenty-four hours of more than one million red corpuscles per cubic millimetre of blood. In profound anæmia the number may fall from five millions to less than one and a half million.

In the liver there is dilatation of the capillaries with endothelial proliferation in the walls; leucocytes and pigment cells lodge in them. Hepatic cells become hypertrophied, and increased in number. There is distension of lymphatic spaces, and sometimes commencement of annular cirrhosis, these conditions tending to become chronic.¹

At the meeting of the British Medical Association at Cambridge, specimens of bacillus malarie were shown by Dr. L. Aitken, of Rome.²

These important and interesting researches, if confirmed, will solve a hitherto unexplained problem.

Dr. Sternberg of the United States Army, has made experiments on the soils, water, and air of New Orleans, and is unable to confirm the views of Klebs and Tommasi-Crudelli. He found that a great number of minute algæ, including bacteria of various forms, do exist on the surface of the swamp mud, and that they could be cultivated in isinglass, which then acquired pathogenetic properties. Some of the organisms found in swamp mud

¹ Lyman in Ziemssen's *Cyclopædia*.

² In the 'British Medical Journal' of December 10, 1881, Dr. MacMunn, of Wolverhampton, gives an account of his discovery of bacillus malarie in the blood of a young African traveller who was suffering from intermittent fever. The blood was taken during the cold stage, and examined with a $\frac{1}{18}$ immersion. The bacillus malarie was seen most distinctly by himself, the patient, and another medical man. The cold was followed by the hot and sweating stage, so that there was no doubt as to the nature of the disease.

and gutter water, and in human saliva, are capable of multiplying within the body of an animal, and the blood, serum, and organs containing them acquire virulent properties. Among the organisms found in swamp mud are some which closely resemble, perhaps are identical with, the bacillus malariae of Klebs and Tommasi-Crudelli. Still, there is no conclusive evidence that these or any other of the minute organisms found in such situations, when injected beneath the skin of the rabbit, give rise to a fever corresponding with the ordinary paludal fevers to which man is subject. The evidence on which Klebs and Tommasi-Crudelli have based their claim to the discovery of a bacillus malariae cannot be accepted, because, in these experiments as in his own, the temperature curve of the rabbits operated on has in no case exhibited a marked and paroxysmal character; because healthy rabbits sometimes exhibit diurnal variations of temperature as marked as those shown in their charts; because changes in the spleen, such as they describe, are not evidences of death from malarial fever, inasmuch as similar changes occur in the spleen of rabbits dead from septicæmia produced by the subcutaneous injection of human saliva; and because the dark-coloured pigment in the spleen and marrow of bone cannot be taken as evidence of death from malarial fever, inasmuch as this is frequently found in the spleen of septicæmic rabbits. He adds, however, there is nothing in his own researches to prove that the so-called bacillus malariae, or some other minute organism associated with it, is *not* the active agent in the causation of malarial fevers in man; and that there are many circumstances in favour of the hypothesis that the etiology of these fevers is connected, directly or indirectly, with the presence of these organisms or their germs in the air and water of malarial localities. He suggests the expediency, as the disease is not of a fatal character, that the *experimentum crucis* should be made on man himself. It certainly seems unsatisfactory to argue from the rabbit to the man, especially in the case of a poison to which, whatever its

nature may be, the lower animals are of doubtful susceptibility.

The researches of Klebs, Tommasi-Crudelli, and others do, however, offer an explanation which appears to correspond with what we know of the mode of operation of malaria. *First*, that it occurs at certain heights, and that it is not necessarily connected with the presence of marshes, ponds, or rivers, nor with the admixture of fresh or salt water, nor with the putrefaction of an organic substance.

Second. That the production of malaria ceases if the air can no longer act on the soil, as when the most pestilential marshes cease to be so if there is plenty of water, or when the air is excluded by any interposing substance.

Third. That a very moderate degree of humidity will produce malaria—some malarial soils, innocuous during hot and dry weather, becoming dangerous after a shower, and also in the case of the upturning of new ground or the cutting down of jungle.

If these views be confirmed by further observation, a great acquisition will indeed have been made to our knowledge, one as important as that which revealed the true etiology and pathology of continued fevers in Europe.

But in our natural anxiety to find a particular origin for the poison or germ, we must not overlook the possibility that the results attributed to the so-called malaria may be due to other agencies—some gaseous emanation, or perhaps an impression produced on the organism from without, inducing disturbance of the innervation, vasomotor action and nutrition, or the autogenetic production of a poison in the body predisposed to be so deranged by peculiarity of constitution, climatic or other influence, of the nature of which we are ignorant. This by analogy is conceivable; as, for example, the origin of influenza, the derangements arising from emotional states, like joy, fear, &c. Some such view has been held by men who find it difficult to reconcile the phenomena and facts with the operation of a specific poison.

Great diurnal ranges of temperature, vicissitudes of climate, fatigue, hunger, exhaustion combined with local causes that predispose to enfeebled health, have been, and are, considered by some as sufficient to account for the phenomena. Surgeon-Major Oldham, of the Bengal Medical Service, attributes them altogether to alternations of temperature in persons weakened by tropical influences. Dr. Lyons, of the same service, says: 'The opinion that intermittent is due to miasm is not applicable,' and Dr. Bellew ascribes fevers in the first instance to chill. All this would imply that malaria as an entity has no existence.¹ Others have expressed similar views, even Dr. Parkes considering that 'some alteration must be made in the prevalent opinion of the action of malaria'; and indeed, there is much in observation of disease in India to support this view—for such extremes are fertile sources of fever; though it is doubtful if they are adequate to produce periodic fevers in those who have not previously been exposed to certain other, *i.e.* malarial, influences. A native of India will get a severe attack of ague if exposed to the chill night-air in the cold season, especially after a hot day; but though it take the form of ague, it may pass away, not to return till he be again exposed under similar conditions.

The most careful examination has failed altogether to detect any chemical product or separate active principle, though it has been shown that the air of marshes contains a variety of products that are absent from the normal atmosphere, *e.g.* excess of carbonic acid, 6 to 8

¹ Deputy Surgeon-General Moore, of the Bombay Medical Service, an able and keen observer, after a long and interesting summary of the facts, says:—'The practice of regarding so many fevers as malarious has tended very much to the retention of malaria as a presumed entity, and as a specific cause of disease, also to its being confused with climatic influences;' and the writer tends to the belief that *so-called* malarious maladies are due to atmospheric vicissitudes rather than to any specific poison, or, in the language of an old Indian colonel: 'You doctors may talk of marsh poison as you like, but my experience has taught me that hot days and cold nights are certain to produce fever.'—*Indian Medical Gazette*, Nov. 1, 1881.

per 1,000 volumes; sulphuretted hydrogen, phosphoretted hydrogen, watery vapour, ammonia, free hydrogen, organic matter, and the microscopical *débris* of vegetable and animal matter. One or other or several of these may be present, but they are not malaria. That some subtle agency in addition to climatic influences is at work, seems probable, but whether it be one or more, we cannot say.¹

This theory of the material nature of malaria is, at any rate, a good working hypothesis, even though we cannot demonstrate its actual existence; and as such only would I receive it, pending more definite information.

A glance at the distribution of fevers in India will show that there, as in other parts of the world, they occur in districts that present opposite characters; and it would be easy to show that they are sometimes absent altogether where marshes and other conditions presumably favourable to their existence abound, and that in arid regions in Africa, Spain, on the sandy soil of Walcheren, in Greece, the island of Ascension, &c., the fevers are often severe.

You will remember Madame de Staël's description of the Villa Borghese, one of the most lovely of the many beautiful places in or near Rome :—

'Tout est là pour la pensée, pour l'imagination, pour la rêverie; les sensations les plus pures se confondent avec les plaisirs de l'âme; mais quand on demande, pourquoi ce séjour ravissant n'est-il pas habité? on vous répond que le mauvais air (la cattiva aria) ne permet pas d'y vivre pendant l'été. . . . Le

¹ Though Mr. Moore says: 'There are quite sufficient recognisable climatic influences from which we require to be protected, without adding an unrecognisable agency under the name Malaria.'—*Indian Medical Gazette*, Nov. 1, 1881.

Again, Dr. C. Gordon, C.B., the distinguished Surgeon-General of the Madras command, says :—'That although in certain localities the presence of an entity, *malaria*, has, and apparently with good reason, been assumed, the precise nature of that entity remains undemonstrated. In other localities diseases admittedly malarial occur in the absence of any possible malaria. This being the case, such diseases, malarial in their phenomena, can only be assigned to climatic and other influences operating in those localities. Hence, interchange in regard to the significance of the expressions '*malaria*,' '*climate*,' '*climatic and endemic influences*.' . . .

mauvais air est le fléau des habitants de Rome—l'influence maligne ne se fait sentir par aucun signe extérieur; vous respirez un air qui semble pur et qui est très agréable; la terre est riante et fertile; une fraîcheur délicieuse vous repose le soir des chaleurs brûlantes du jour; et tout cela c'est la mort.'

Klebs and Tommasi-Crudelli would say that death was in the bacillus; Oldham that it was in the cold night after the hot day.

MacCulloch believed in malaria from a variety of sources: the surface of damp ground; still, stagnant waters, from the lake to the smallest pool, the dung-heaps and pools at the doors of farmhouses and cottages; from sewers and ditches; from the mud left by the recess of the tide, and at the mouths of rivers; the minute marshy and swampy spots in low situations near woods and on roadsides, or small spots of coppice and brush-wood in England. He mentions a case in the West Indies where a number of men were seized with fever from contact with some ground which, by the removal of boxes, became exposed to light and heat. From the mere malaise of the disordered health which many people feel in certain localities that are damp and ill-ventilated and built on damp ground, to the severest forms of remittent, he ascribes them all to malaria. We are not wont to call it malaria unless it produces periodic fever, neuralgia, or cachexia; but we do not unfrequently find people recover from a depressed state of health with anomalous symptoms on removal from a damp locality, and in this sense we may call it malaria. The depressed health that results from exposure to impure air from drains and sewers (I mean irrespective of specific poisoning) is of the same character, and the ill-effects of such localities on persons who have previously, it may be long before, suffered from tropical fever, are not uncommon. I have seen cases, where each autumn brought back vague indefinite symptoms of malarial fever, or neuralgia, and rheumatism, quite restored to health by quitting the locality at that time of the year; and lately we read of

malaria from flower-pots. I have heard of it from watering a flower-bed in India, and of an insidious disease described as 'drawing-room malaria,' arising from the presence of living plants in the hot, damp air of rooms.¹

Malarial fever is the origin of most of the diseased conditions I have to deal with in connection with Indians who come home for the benefit of health, or who appear before me officially at the India Medical Board; but I occasionally see persons who present the same appearances, and, indeed, actually suffer from periodic fever, who have never been in the tropics, and for whom it is not easy, even by a stretch of imagination, to find a malarial origin for the disease.

The existence of malaria as a material peculiar entity has not yet been demonstrated, and it is still asked if such a thing exists? Dr. Macnamara says, in his work on 'Himalayan India': 'What is meant by the term malaria? Does it simply express the result of certain climatic influences, or does it imply the existence of something more material as a poison?' Is that which we call malaria the sum of the operations of the various conditions of climate and place by which we are surrounded? It may be so; there are circumstances connected with its

¹ We no longer have much cause to fear the 'black deaths' and 'sweating sicknesses' which decimated whole towns or provinces in the Middle Ages; but we have surrounded ourselves with dangers unknown in a less civilised community. One of these is an insidious disease lately made known and described as drawing-room malaria. The atmosphere of a hot room in which many living plants are kept has quite recently been found to be impregnated with a moist vapour arising from the earth in which these plants are rooted. The soil from which they derive their sustenance is generally rich in organic matter. The required proof has come from Russia, where the sitting-rooms in winter are kept habitually at a very high temperature with little ventilation. Professor von Eichwald was consulted as to the health of a lady who, though living in a healthy spot, exhibited all the symptoms observable in those who inhabit marshy places. The usual remedies, consisting chiefly of quinine, were applied with success; but as often as the lady, after recovering from an attack, ventured into her drawing-room, the same symptoms persistently reappeared. The doctor ordered the removal of the numerous ferns and plants which filled the drawing-room, and the complaint which had been so obstinate was found to disappear at once.—*Globe*, Dec. 31, 1881.

action which are difficult to reconcile with a parasitic origin, and for the present our attitude with respect to this point, much as we may wish that it should prove all true, must be one of reserve. But who that has followed the progress of pathological investigation during the last quarter of a century would venture to assert its impossibility or its improbability, or that in such researches as those of Pasteur, Burdon Sanderson, Lister, Greenfield, Koch, Klebs, Tommasi-Crudelli and others, we may not find a complete solution of the problem? ¹

¹ In a valuable paper read to the Epidemiological Society last November, Dr. Henderson says, speaking generally :—‘ Are these organisms the causes of the various processes, or are they merely associated or epi-phenomena, or are they merely accidental complications of the processes? In helping our decision we have three sets of facts which tell in favour of a causal relations.

‘ 1. The organisms in either the blood, some of the tissues or organs, or in morbid fluids in animals suffering from the disease, whether developed spontaneously or after inoculation.

‘ 2. Blood or fluids containing them or their spores transmit the disease when inoculated ; when they are absent, or have been artificially separated, inoculation gives no such result.

‘ 3. They can be cultivated, still preserving their specific form, outside the animal body, and after five, ten, or fifteen generations, reproduce the original disease when inoculated ; all three kinds of evidence are found in the case of anthrax, some varieties of artificial septicæmia, pneumo-enteritis of pigs, and fowl cholera ; and we have as strong reasons for believing that the organisms are the cause of the diseases in question, as for holding that the ingestion of cysticerci in measly pork or beef is the cause of the tape-worms we subsequently find in the intestines.

‘ With regard to relapsing fever certainly, and probably variola, vaccinia, erysipelas, and diphtheria, and possibly enteric fever also, if Klebs’s observations are correct, we have only the first and second kinds of evidence ; hence, though there is a strong probability that the organisms do cause the diseases, we cannot look upon the question as absolutely settled till experiments in artificial cultivation and retro-inoculation have been made.

‘ Yet, though trichinæ have not been “grown pure” in artificial media, no one doubts that their development and migrations into the tissues is the cause of the general disturbance of the system known to us by the name of trichiniasis. In the case of scarlatina, ague, and leprosy, we have as yet no positive evidence of the nature of the organisms described as afforded by inoculation and cultivation in the first and second class of diseases. While, therefore, from the analogy of other diseases, and from the gradual elucidations of one point after another in their etiology, we may hope for

Facts and Modes of Action of Malaria.

Briefly to summarise the facts about this so-called malaria and the methods by which it acts—whatever it may be—it seems to be greatly influenced by local and climatic conditions, its activity increasing generally with proximity to the equator. Absent from the arctic, feeble in the temperate, it becomes most concentrated in tropical regions, though there are parts of Asia in North India, as well as of Europe, where it is most active. Though prevalent in low-lying, marshy, or water-logged ground, or on soil drying up after rain—as in the Himalayan Terai, or the Sunderbunds of Bengal, in Assam, or on land that is rendered damp by damming of watercourses and interrupted drainage or saturated subsoil from irrigation (as has been remarkably illustrated in the last few years in the division of Burdwan, which has suffered severely from a low form of malarial fever)—it is also found on dry, sandy, or rocky ground, where there is little or no moisture or vegetation of any kind. Of this there are many examples in Europe and in India. But still, water seems to be the prime causal agent, if not on the surface, in the subsoil, especially when *stagnant* and near the surface. Water seems not only to determine the generation of malaria, but to hold it in solution. The natives of India attach little importance to atmospheric states, but firmly believe that the water of pools and tanks, or even of streams flowing through certain jungles or marshy places, is charged with fever poison; and many believe that the milk of buffaloes or cows fed in these places has the same property, as I have myself heard natives in the Terai assert. Malaria is more active near the surface of the ground, and in valleys, hollows, deep, dry ditches or moats, low alluvial soils, old tanks filled with refuse, silted-up beds of rivers, dams across streams, and obstructed

future settlement of the question, we are not at present in a position to decide whether they are causes or consequences of the maladies in which they occur.'

watercourses. Decreasing in energy with height, it ceases to exist above certain altitudes, variously given at from 1,500 to 5,000 feet. How high it permeates the air above the sea-level surface is not known, but it is certain that the top of a hill, even the upper room of a house, is less dangerous than the ground-floor. It occurs in some of the hill stations of India, but as there is constant communication with the plains and valleys, this may be the result of importation. It moves like mist and rolls up the hill-sides, nay, overtops those of a certain height, and may be dissipated by or travel with the wind, probably some miles, but no one can define any particular limit—though it proceeds to a greater distance over land than over water, especially salt water, which is supposed to have the power of absorbing and retarding it. Crews of ships lying at a considerable distance to leeward of a malarious shore have been affected by the off-shore wind, and it is said that ships have generated it from certain cargoes of green wood, coals, or other vegetable matter; that they have evolved miasmata from rotting timber or bilge, which have developed fever of great severity. Steeping of hemp, jute, and indigo, or other vegetable matter, has had similar effects. Villages and camps have been affected when to leeward of swamps, even at a considerable distance. It is said that burning fires, smoke, or a belt of trees will arrest its progress, that it clings about trees—hence the danger of sleeping under certain trees—that the growth of trees will destroy or prevent it, and that some, such as the eucalyptus for example, have a special antagonistic power; but there is probably nothing more in this than the rapid growth of these trees which forms them quickly into plantations. A screen of gauze or muslin is said to be protective, and that a mosquito curtain will keep out malaria as well as insects. It is not generated where the diurnal range is below 60°. A very high temperature does not always cause, but may even appear to prevent it, though the other necessary elements seem to be present. Malaria is more

active at night than in the day—more likely to affect those who are exposed to it at that time, especially during sleep, and more especially if lying on or near the ground. It affects the weak sooner than the strong, those of a phlegmatic, lymphatic, or melancholic, rather than those of the sanguine or nervous temperament; the sickly and ill-fed, before the robust; it spares no age; new-comers are more liable to suffer than those who have been acclimatised—it affects all races. The natives of India suffer greatly, but it would appear that the negroes on some parts of the coast of Guinea acquire a tolerance which has been referred, possibly without sufficient reason, to the colour of the skin.¹ There are certain tribes in the Terai and other forest districts of India which acquire some immunity; the non-Aryan races, such as inhabit Assam, suffer, it is said, to a greater extent from malarial disease than the Aryans in the same province. The Tharoos² live where it would be death to others, but even they are not altogether exempt.

Malaria is very intense in the Terai, which is the belt of low, swampy, forest ground at the foot of the Himalaya mountains, where the porous soil has a substratum of clay by which the water is brought and retained near the surface, and where there is dense vegetation and a high temperature. It is so also in certain jungle districts, and water-logged land, and where the tides encroach; in the river valleys, the deltas, and at the debouchures of rivers; near rice and other cultivation in some stages, though the danger from fresh rice cultivation is probably

¹ From idiosyncrasy the negro appears to be proof against endemic fevers, for to him marsh miasmata are in fact no poison; hence his incalculable value as a soldier for field service in the West Indies. The warm, moist, low, and leeward situations, where these pernicious exhalations are generated and concentrated, prove to *him* congenial in every respect. He delights in them, for he there enjoys life and health, as much as his feelings are abhorrent to the currents of wind that sweep the mountain-tops, where alone the whites find security against endemic fevers.—FERGUSSON'S *Notes*, page 204.

² The Tharoos are descendants of an Aryan race. They lost caste from drinking, and breeding fowls.—Sir S. ELLIOTT'S *Races of India*.

exaggerated; and in such localities as the Sunderbunds of Bengal, where the alluvial mud is covered with dense jungle and frequently washed by the salt water—the jungles lying at the foot of hill ranges and along the sea-coast where salt and fresh water mingle, and where organic matters decompose amid moisture and heat. It is scarcely less active on high and arid sandy ground, as in the Deccan, Sind, Bikaner, Peshawur, the Punjab, Bhawalpore; but even in these localities subsoil, damp, and organic matter—for there is always some—appear to be at the bottom of it. Yet there are places, it must be admitted, which seem so dry and devoid of vegetation, and with the water at such depths from the surface, that it is difficult to believe that the explanation holds good—as it probably does—for though there may be no great quantity of water, the subsoil is impregnated by a certain amount of stagnant moisture, which is probably the worst of all. It often appears with great intensity—after excavation and turning up of soil—in land that has recently been broken up, or that has recently been denuded of jungle; whilst, on the other hand, cultivation, draining, and cropping, seem to diminish or destroy it. The worst malarial dysentery that I have ever seen followed the clearing of some jungle during the last Burmese war. There are localities in India now comparatively healthy that were formerly dangerous! Malaria is at its worst in the drying-up season after the rains and the beginning of the cold season; in the dry, hot weather, and during heavy rains when the ground is covered with water, or when the land has been for some time cultivated and populated (compare Calcutta of 1880 with Calcutta of 1780), or covered with trees or even fresh turf, it is less severe. Certain characters of the soil seem to favour its production. Sandy, porous ground, with a substratum of clay, soil containing mineral or organic matter, mixed alluvial deposits, volcanic, rocky, sandy, granitic soils or surfaces, have been thought to favour it, but it is impossible to ascribe it to any particular soil.¹

¹ Fergusson says (page 198, *Notes and Researches*):—‘Endemic fevers

The low-lying swampy ground of the Concan, and the dry arid sandy plains of Marwar, are contrasted by Moore; they are very different, yet malarious fever prevails equally on both. Rice cultivation is by some authorities considered most productive of malaria. Martin and others say it is not so. I am inclined to think that the fresh-growing young rice is innocuous, but that at other seasons the ground on which it grows may be malarious. Pringle and others thought salt marshes insalubrious. Jackson and others did not find their neighbourhood less healthy than other localities. Some severe remittents occur in the vicinity of the salt-water lake near Calcutta.

If, says Moore, salt marshes are deleterious, the neighbourhood of the Sambur lake in Marwar should be deadly; and if marshes overflown by the sea are injurious, the neighbourhood of the Runn of Kutch should be an example. But the fevers in these localities are not more malignant than in other parts of the country.¹ Some have thought that saltiness and alkalinity in the soil conferred

cannot be generated either from aqueous or vegetable putrefaction, singly or combined. It emanates from the shores of the purest streams, wherever they have been flooded during the rains, through want of confining banks, and it is absent from the most putrid waters. It must be impossible that healthy living water, which from its current is in a perpetual course of being refreshed and renewed, can ever by any degree of solar heat be brought into the state of morbid miasmata, and the evil must therefore reside in the half-dried and drying margin, for the swamp is no more than this margin rolled up under another shape, and it must be brought into the same degree of dryness before it can produce any morbid effects.

‘ . . . We may infer that the poison is produced at a highly advanced stage of the drying process. But in the present state of our knowledge we can no more tell what that precise stage may be, or what that poison actually is, the development of which must necessarily be ever varying according to circumstances of temperature, moisture, elevation, perfiation, aspect, texture, and depth of soil, than we can depict and describe those vapours that generate typhus fever, small-pox, and other diseases. It is from the drying margins, and never from the body of the lake or pool, that it emanates.’

¹ Moore also quotes well-known examples:—‘ In Australia, New Zealand, America, and North Carolina and Virginia, there is plenty of ague. Yet in the great dismal swamps on the frontier of Virginia it is never seen. So in Ceylon, Singapore, Malacca, and elsewhere, there are regions with

immunity. With reference to the occurrence of malaria on rocky surfaces, Dolomieu calls it 'la maladie du granit' from the prevalence of malarial disease on certain rocky sites. This has been attributed to the presence of fungi growing in crevices amid disintegrating granite, but the amount is so small that it can hardly be supposed to generate malaria. Mount Aboo, in the Aravulli hills, is such a locality, but, as Moore says, 'there are quite reasons enough why fever should prevail there at certain seasons, without calling in the theory of "maladie du granit."'

Heyne, in a paper on the Hill fevers in India, shows that fevers prevail where granite rocks and iron stone are found in large quantities, and attributes the disease to magnetic or electric fluid disengaged in excess. Martin thought ferruginous hornblende might be the cause. Volcanic soils and exhalations were supposed by Sir W. Napier and Dr. Parkin to cause epidemic fever in Sind. Sulphurous vapour has also been suggested as a cause. Kuler attributed the endemic fever to limestone rocks. Gordon says: 'Fever may occur on rocks, and on the detritus of rocks, as at Gibraltar, Malta, Ascension, the Ionian Islands, Hong Kong, and Cape Coast Castle, when the rock is rotten, and gives out vapours as the sun falls on it after rain.' He tells us also that 'in America near the Orinoco, malarial diseases are described as occurring in localities where there is no malaria as such.' At Port of Spain in Trinidad, W.I., the residents enjoy comparative immunity from fever, though the place is surrounded by a swamp, and yet the same persons, if they take up their abode for a single night in La Vantile hill, in the immediate neighbourhood, overlooking the Bay of Trinidad, suffer from fever in its severest form. 'It is on record that at Baïe the French army suffered very

marshes exposed to great heat and atmospheric humidity, and in localities shut in by mountains, yet are exempt from malarial fever. In Mexico the Lakes of Tescudo, 25 miles area, composed partly of fresh partly of salt water, with a clay bottom, part of which is often laid bare as the result of evaporation with a temperature of 120° to 140° Fahr., and yet malarial fevers are rare.'

severely from malarial fever, although malaria as an entity seemed non-existent.' So at Hong Kong, and Cape Coast in Africa, climatic fevers are severe and deadly, though the soil is dry and hard in both places; vegetation is spare at Hong Kong—at Cape Coast it is dense, though not so in the same sense as in an Indian jungle. At both the underlying rock consists of decomposing ferruginous granite, and at both the alternations in meteorological conditions are great and sudden, and malarial diseases prevail. In Sind and the Punjab the soil is mostly sand or alluvium on clay. At Kurrachee it is magnesian limestone, yet malarial fever, neuralgia, and cachexia are common.

I need not cite more proof that malaria may occur under very opposite conditions, and that it is impossible to assign it to any one of them. The question arises—may it not be the outcome of several causes which must co-exist before the effect is produced? Atmospheric vicissitudes, heat, damp, telluric exhalations, impurities of air, water, and neglect of personal hygiene; not one, but perhaps many or all, may have to come into operation before periodic fever is produced.

Indian experience, however, supports the view expressed long ago by Pringle, that the chief determining cause is stagnant subsoil water, under certain temperatures; for when such water moves however slowly, the evidences of malarial poisoning are less marked. To the stagnant water must be added a certain combination of air and decomposing organic matter. What part may be taken respectively by vegetable and animal matter in the production of malaria is not known, but it is impossible to conceive of miasmata arising from organic decomposition in a tropical marsh that is not a mixture of both; for low forms of animal life teem among the vegetation in such places, and the slime and ooze of a swamp drying up must contain quantities of animal matter, dying and dead. This may perhaps explain the more virulent character of some miasmata, and account for

varying phenomena. The miasmata given off from rocky soils, having less of the animal element, may account for differences that characterise the fevers of those regions. We must also bear in mind the influence of local conditions in determining the activity of malaria. The late Dr. Melier, in his report on the *marais salins* of France, says of the fishponds of Lindre Basse, that in the first year of fish cultivation, when the pond was half-filled, intermittent fevers prevailed. In the second year, when full of water, enteric fever prevailed; and in the third year, when dry and cultivated, carbuncular affections prevailed. These diseases succeeded one another as regularly and invariably as the different states of the ponds for a period of sixteen years. But a change in the rotation in 1848-49 altered the order of succession of these diseases. Here climatic conditions seem to have been the same throughout, but the local conditions were altered, with a corresponding alteration in the disease.'

Taking all the facts together in order to produce the effects called malarial, you must, Dr. Sutherland observes in a letter addressed to me in November 1881, 'have water, temperature, and organic matter. If the matter be of vegetable origin, you may, according to its nature, amount, and rate of decomposition, have various types of intermittent fever, passing into remittent in aggravated cases. If it be of animal origin or of animal and vegetable origin conjoined, you may have remittent or continued fever, Bulam, yellow fever, or enteric fever. But the personal predisposition and climatic causes must always be taken into account.'

Action of Malaria on the Lower Animals.

With reference to the influence of malaria on the lower animals, I am indebted to Professors Fleming and Axe for the following most interesting information. MacCulloch also describes its effects on animals.¹ Professor Axe says :—

¹ MacCulloch *On Malaria*.

‘With reference to the influence of malaria on the lower animals, I am sorry to say our data are few and unreliable. I am not aware that periodic fever does at any time prevail in the lower animals of this country from malarial influences. Acute splenic enlargement is, as you will be aware, common in “anthrax” disorders, septicæmia, and pyæmic infections; but I am not aware that it has any enzootic existence in those localities where malarial disorders are known to prevail in man. I may mention that in the low-lying marshy districts of Lincolnshire, Cambridgeshire, and some parts of Scotland, cattle are frequently the subjects of circumscribed cellular abscesses in the vicinity of the throat and neck, which may possibly have some connection with malarial emanations, but we are much in want of precise data on the subject. The question is a highly important and interesting one, and deserves the fullest investigation. Should an opportunity offer, I will make it a matter of special study, and you shall know the result.’

Professor Fleming says:—

‘The occurrence of malarial fever in animals has been often denied, but a host of observers, in every way trustworthy, have testified to its existence in nearly every part of the globe. Those to whom I can just now refer as witnesses are Carlo Ruini and Lancisi, in the sixteenth and seventeenth centuries; in the eighteenth century, Kerstnig, Waldinger, and Veith; in this century, Damoiseau, Rodet, Liégeard, Hamont, Clichy, Blanc, Hering, Spinola, Hertwig, Dressler, Delwart, Legrain, Bertacchi, and other veterinarians of repute. Lessona, a distinguished Italian veterinary surgeon, has frequently observed it affecting horses, cattle, and dogs in Sardinia. Blaise has seen it in the same animals in Algeria; Hamont also in Egypt; Bertacchi in the Pontine Marshes; Hildreth in America (affecting dogs). Dupuy and Cleghorn assert that sheep are greatly predisposed to it. Jenisch has seen it in the pig, and Czermak states that he has noticed it not only in dogs, but in monkeys also.

‘The attacks resemble those of paludal fever in mankind, and their duration is usually brief—from half-an-hour to six or seven hours, after which the animal is apparently easier; though if closely observed a certain degree of *malaise* will be noted—as frequent yawning, saffron-tinted mucous membranes (this is most marked during the attack); tension of the abdomen, which is often painful on pressure in the region of the liver; often there is gastric disturbance, and in the dog, vomiting. All the types of the fever have been witnessed, quotidian, tertiary, &c.

‘When the disease continues for some time, a cachexia appears to be developed, with localisation of lesions in certain organs, the most notable of which are those of chronic congestion, particularly of the spleen, sometimes the liver, in other cases the lymphatic glands.

‘It is extremely difficult indeed to diagnose the presence of neuralgia in dumb animals. I have only seen it once, for certain, in a dog.

‘Veterinarians have long noticed that domestic animals living in malarious regions have the paludal cachexia, and that in them intermittent fever often complicates or accompanies other maladies, sometimes even occurring after operations or accidents. Anthrax or charbon was, until the discovery of the *Bacillus anthracis*, believed to be malarial. The horse-sickness of South Africa is even now believed to be malarial, as people suffer severely from intermittent or remittent fever when and where it prevails; but I am now quite convinced that it is only anthrax, as it is inoculable, and I have found bacilli in the blood. The terrible “Yaswa” of Russia, which kills enormous numbers of horses, cattle, and other animals, as well as (by transmission) people, is only witnessed in malarial regions, but it also is only anthrax.

‘I may add that in 1861 I had several cases, among horses in North China, of what I considered well-marked intermittent. I forgot to mention, in connection with the horse-sickness of South Africa, that dogs suffer in the

malarial districts of that country from a disease which is evidently malarial. It is more particularly noticed in the Valley of the Limpopo, and away north to the Zambesi. It is known to the Boers as the "hond-ziekte," or dog-sickness, and is very fatal. The liver appears to be the organ chiefly involved, as the mucous membranes are deep-yellow in colour (*geel bec*, or "yellow-mouth," of the Boers), the abdomen is enlarged, there is jaundice, constipation, and high temperature.'

Distribution of Malaria in India.

I have endeavoured, in a map,¹ to give an idea of the geographical distribution of fever in India, and it will be observed that no part of the Empire can be said to be exempt, though in some, such as the hill stations above 4,000 or 5,000 feet, malarial influences must be practically non-existent. Those parts that are coloured lighter than the rest represent the feudatory States and Bhawulpore, where there is no registration, or none such as to enable one to estimate accurately the extent of malarial influence. Bengal, Bombay, North-West Provinces, Oude, and Assam, are numbered 3. Assam in many parts, no doubt, should be more, but 3 represents its average; Madras, 2; Burmah, Scinde, and Mysore, each 1. Of course this is a mere approximation, for any attempt to represent the incidence of malaria in this way must be simply a combination of the results of registration and personal experience. Registration, confessedly imperfect, is rapidly improving; and it shows that periodic fever prevails everywhere, yet is not sufficiently complete to define clearly the actual intensity in any region. If, for example, one were to be guided by registration alone, it would show some provinces, such as Assam and Bengal, to be much less affected than others that are well known to be healthier. The map, for which I am indebted to Dr. Sutherland and Mr. Trelawny Saunders, is constructed

¹ Exhibited at the Lecture

from the returns on the principles described. The different shades of colour indicate the relative intensity in different provinces. The most unhealthy, such as the Terai, is marked as 7. The Doab, where irrigation is prevalent, as 6; Berar, 5; Central Provinces, 4; the others as before mentioned. The annual rates of fever mortality for five years, 1875 to 1879, are as follows:—

Bengal	12 20 per 1,000 approx.
N. W. Provinces, Oude, including Terai	30·78 " "
Punjab	23 75 " "
Central Provinces	24·45 " "
Berar	27·99 " "
British Burmah	11·09 " "
Madras	16·31 " "
Bombay	22 97 " "
Assam ¹	8 87 obviously too low.
Mysore, 1879	10·86 per 1,000 approx
Coorg, 1879	19 71 " "

DR. SUTHERLAND.

¹ *Sanitary Report of Assam for the year 1880.*

FEVERS.

EXTENT OF PREVALENCE AND FATALITY OF FEVERS.

Year	Number of Deaths
1877	18,715
1878	17,398
1879	26,731
1880	31,272

The number of deaths registered under the term 'fevers' was 31,272, and far exceeded that of any previous year. The table exhibits for the past four years the number of deaths registered throughout the province under this head. The total registered death-rate appears to increase yearly in this direction. March and April register the lowest numbers; October, November, December, the highest. The disease increases with the rainfall, and continues in severity after the rains and in the cold weather. Even if we could accept the total number of deaths registered as correct and due to malarial causes (marsh-miasma), we should not be surprised. Assam possesses all the factors of malaria in the most concentrated form possible. The people live in the midst of marshes, surrounded in many parts by dense jungle-grass from twenty to thirty feet high; they have no artificial drainage, and the subsoil water-level is nearly always close to the surface of the ground. Everywhere the climate is very humid; everywhere, almost, there is rank luxuriant vegetation side by side with vegetation

General features of distribution only are represented. Many additions might be made in certain localities, such as river-valleys, deltas, swamps, jungles, irrigation districts; and along all the submontane districts loose gravelly land with a clayey subsoil might be represented by a darker shade of colour, but this would involve an amount of detail that is not practicable now.

The other maps, to which I would call attention, are:—One representing fever-prevalence among British troops; one of the rainfall, in which the amount of rain, from 4 inches to 300 or more in the year, is represented in dark shades; also maps showing the chief divisions of India; the hill ranges; rivers, deltas; water-sheds; table lands; low lands; and littorals.

It may be asked, is malaria always the same, or does it only differ in concentration and activity according to circumstances of season and place under which it is generated? Is that which causes jungle fever, bilious or ordinary remittent, simple ague, cachexia and neuralgia, one and the same, or is it of different kinds? Is the malaria of the Terai the same as that of the dry soil of Sind? Unless it be proved that malaria is due to organisms, we know nothing of its essential nature. The cause, therefore, of the varying effects must also be a matter of speculation, though the tolerably constant recurrence of certain effects corresponding to certain recognisable conditions, suggests the probability that the decaying and decomposing; and both air and water cannot be other than impregnated with the concentrated poison of malaria. The huts are dark and damp, and embedded in the midst of high growths of bamboo, betel-nut, and plantain. In many of the districts whole areas are flooded for months. Under such conditions we cannot wonder that malarial poisoning should, year by year, be the cause of a high death-rate. This year the highest registered number yet recorded from this cause is 60 per cent. of total deaths.

There is no mention in either of the three reports of any unusual sickness from 'fever,' and both the Civil Surgeon and Deputy-Commissioner of Sylhet remark on the healthiness of the district during the year. The registered total death-rate from fevers has risen in Sylhet from 4,140 in the year 1877, to 13,937 in the present year. Presumably, this apparently increased death-rate arises from improved registration.

poison is only one (modified by circumstances) which, proceeding from drying-up swamps or marshes under the influence of atmospheric conditions, including oxygenation, will most probably cause simple ague; though it is possible that the disease from the same marsh may be more intense under other atmospheric states. In this way, what are called unhealthy years in the same district may take their origin; but in all such cases there are two factors to be considered, viz. the malarial cause and the constitutional stamina of the population exposed to its action.¹ Excluding individual predisposition, we are probably justified in assuming that different degrees of intensity or concentration of the miasm produce the different effects.

Thus, the malaria of the Terai or Sunderbunds causes dangerous remittent; that of the general surface of Bengal, ordinary ague or milder remittent, or malarial cachexia; that of colder climates, a variety of indefinite complaints rather suggestive of general ill-health than any specific disease—a condition which is insisted on by

¹ Dr. Eccles says:—‘A battalion of Arabs who, before arriving in Thessaly, had inhabited a desert region in Syria, with no vegetation except at great intervals, presented a large majority with the characteristics of people who had suffered from ague: sallow complexions, yellowish sclerotics, wrinkled skins, unnaturally protuberant abdomens, enlarged spleens, and a general look of premature old age, with the peculiar dulness of the mental faculties. This type varies with the dampness of the soil and the stagnation of the atmosphere. In the plain of Larissa, where the soil is dry and under cultivation, but subject to heavy rains at certain seasons of the year, followed by great solar heat, it will be found that the fever produced is generally of a comparatively mild type, probably quotidian, while pernicious or remittent will not occur except as an intensification from excess or want of ordinary care, or neglect to arrest the course of the disease; whereas, when a patient not predisposed to ague by a previous attack has been exposed to the effects of malaria arising in a damp marshy locality, or in alluvial soils, such as the banks of a river subject to overflowing, he will suffer from a more severe form of the disease than if he had been attacked in a cooler and drier region.’

He has seen ‘remittent occur *de novo* in a swampy locality where very warm days are followed by cold, damp nights; while on the higher grounds and on the plains at a distance from any apparent miasm, the disease has invariably been, in the first instance, mild and benignant in type.’

some at the present day, whilst MacCulloch attributed half the ailments in our country to it, as arising from every pool, pond, ditch, or plantation in England, and found the causes of ill-health in these local developments of malaria.

Are the symptoms of depressed health seen in some damp localities in England referable to a malaria?—most probably they are. Instances might be cited to show that ditches, drains, moats, pools, newly-watered ground, ships' holds, bilges, or ground newly turned up, give rise to fever in various degrees of intensity. The writings of Pringle, Fergusson, MacCulloch, and others furnish instances.

Contrary to what might be expected, natives suffer more than Europeans in India, no doubt on account of the unfavourable conditions under which they live. The Europeans are, for the most part, well housed and fed, and guarded against insanitary influences. The natives are much exposed to the action of malaria, poorly fed and badly housed. Fifty years ago Europeans died of fever at the rate of 13·25 per 1,000; in ordinary years now less than 3 per 1,000 die of this cause, and there is reason to believe that the rate will be still further reduced; in natives it is still high, being nearly 25 per 1,000.¹

¹ In the British troops in the Bengal army the deaths from all fevers during the sixteen years, 1830 to 1845, amounted to a ratio of 13·25 per 1,000.

In the year 1875	.	.	.	2·77 per 1,000.
„ 1876	.	.	.	2·46 „
„ 1877	.	.	.	2·21 „

In 1878–79 there was more fever from the famine and the Afghan War.

The army of India, five years, 1871–75 :—

Deaths from all fevers	.	.	.	2·81 per 1,000.
„ „ 1876	.	.	.	2·41 „
„ „ 1877	.	.	.	2·16 „
„ „ 1878	.	.	.	5·07 „
„ „ 1879	.	.	.	6·55 „

The rates for 1878–79 were part of the great fever epidemic which swept over India and occasioned enormous losses to the civil population, and the rates of these years include the Afghanistan fevers and also those of

Seasonal Prevalence.

What Celsus, quoting Hippocrates, said of Europe, applies to India, ‘“ Igitur saluberrimum ver est, proxime deinde ab hoc hiems ; periculosior æstas ; autumnus longe periculosissimus. Ex tempestatibus vero optime æquales sunt sive frigidaë, sive calidaë ; pessimaë quæ maxime variant. Quo fit ut autumnus plurimos opprimat.”’ In the hot months from March till June, fevers are more of a continued and ardent type ; and are apt to prove dangerous and fatal from cerebral complications, and seem to be pathologically linked with ephemeral fever on the one hand and insolation on the other, differing, as I believe, only in degree. They are due chiefly to over-heating, and are liable to be modified by malarial influences. In the rainy season, and on to September or October, forms of intermittent, remittent, and continued type occur, while there is also a tendency to dysentery and bowel complications. After the rains, during the evaporation and drying up of the wet ground, fevers of the malarial type become prevalent. When the cold season sets in they also recur, in many cases re-excited by the cold, especially in the early part of the cold season, before the system becomes habituated to the change,¹ the weak, anæmic, or exhausted

troops on the march. Of the civil population of India there died from fevers :—

In 1877	.	.	.	2,504,493.
„ 1878	.	.	.	3,247,271.
„ 1879	.	.	.	3,564,035.

¹ Report on Sanitary Measures in India, India Office, 1877, 1878, to 1879. The Sanitary Commissioner of the Punjab (Dr. Bellew) made a careful inspection of several towns and villages with a view to investigate the causes of the widespread prevalence of fevers in that province.

An examination of the registers showed that by far the largest portion of the mortality occurred within and up to fifteen days of illness ; also that out of 15,432 deaths, no less than 3,676 were in children from a few days old to five years of age.

Fever-deaths are registered without distinction of type or form, but practically there is no great difficulty in estimating roughly the relative frequency of periodic or malarious fevers to those of continued or specific kind.

An examination of the eight years 1869-76 brought out the fact that

suffering, as has been described by Twining and other writers. That such changes revive fever in those who have previously suffered is well known, for who has not heard old Indians say 'the cold had brought on a return of former Indian fever, from which they had not suffered

the fever mortality of the province invariably rose by a sudden bound of several thousand deaths in the month of September after the fall of the monsoon rains, whether those rains fell from June to August or from July to September. The higher mortality continued through the autumn months, and declined with tolerable uniformity through the winter and spring months, till the minimum was reached in March and April, after which there followed an alternate rise and fall till the next monsoon.

An increase in the annual aggregate fever mortality was found to bear no fixed or definite relation to the increase in the aggregate rain-fall.

The fevers which follow, with increased prevalence, the periodical rains are mostly of the malarious kind. The other kinds of fever are also most common in the winter and spring months, but they prevail more or less through all the seasons of the year; which is indeed the case, though to a less degree, with malarious fevers too.

Dr. Bellew concludes that canals exercise a direct influence upon the prevalence of fevers in the direction of their greater development and fatality, through the increased humidity of the soil and atmosphere producing chill.

He further shows that the prevalence and virulence of these malarious fevers are very largely influenced by insanitary conditions of locality and soil.

'The remedy,' he adds, 'must be sought for by a special study and investigation of each case separately, and whatever other provisions should be deemed necessary as the result of such inquiry, a good drainage and an efficient conservancy must under any circumstances and in all places be insisted on so far as practically attainable, as the prime and indispensable requisites, not only for the preservation of a wholesome water-supply and pure breathing air, but for the maintenance of the sound health and comfort of the community at large. . . .

'An epidemic of fever of the so-called malarious kind, which, in the first instance, was produced by atmospheric agencies acting both directly and through the medium of the soil, may also be sometimes, and is, as a matter of experience, very quickly transformed by the action of local sanitary defects into an epidemic of quite a different kind, in which fevers of a continued and distinctly specific type predominate. . . .

'The over-crowding, insufficient food, want of clothing, and generally filthy surroundings of the poor classes of this country, and they form a very large proportion of the general population, are the chief causes which combine with the sanitary defects above mentioned to produce among them, not only in the towns and cities, but also in the villages, the several specific fevers, viz. enteric, typhus, and yellow, from which they so largely suffer both in the epidemic and sporadic form.'—BELLEW.

for years'? Two of the severest attacks of ague I have had as reminiscences of remittent of former years, were caused by getting into a bed with cold linen sheets, and by a douche of cold water after the manipulations of the hair-cutter.

Effects of Malaria on Man.

In addition to those effects for which malaria is directly responsible, a diathetic condition seems to be established, which modifies other diseases. The experience of Indian medical officers will confirm this, for periodicity complicates nearly all disease in India. The effects of malaria are indeed most protean in form, not only in its own definite and well-marked pathological processes, but in simulating others, from the stupor of typhus, the collapse of cholera, the high temperature of insolation, the sickness from an irritant poison, to the convulsions of epilepsy or of dentition, all of which may occur in the pernicious forms. It induces anæmia and general cachexia, with structural changes in the liver, spleen, or other viscera, neuralgia, asthma, and various other symptoms of disturbed innervation and sanguification; and, as I have before said, appears to be in close etiological relation with dysentery, cholera, diarrhoea, beri-beri, hydrocele, elephantiasis, bronchocele, and hepatic disease.

Whatever its nature may be, the action of malaria on the human economy is very striking. It affects the central nervous system, causing disturbance of vaso-motor action, expressed in rhythmical paroxysms of fever and congestion of the abdominal viscera, which become either permanent or periodic in recurrence, and may pass on to structural changes in the liver and spleen, or intestinal mucous membrane. The nervous system also is prone to suffer, and neuro-asthenia, neuralgia, or asthma may result. It confers a special character on other diseases, and sets up, as it were, a malarial diathesis. No one can have resided long in a malarial climate, like Assam, without observing the broken-down, cachectic, deteriorated aspect of the

people, who, although they may never have had a single attack of fever, do not feel ill, and would resent the imputation of being considered so, are yet victims to the insidious action of the poison, and present evidences of anæmia, degenerate tissues, and chronic visceral disease.

In my next lecture I shall describe intermittent fever as it occurs in India.

LECTURE II.

IN my first lecture I gave a brief description of the physical characters of the country, people, climate, and prevalence of fever in India, and of the theories regarding the nature and origin of malaria. I now propose to consider the types of fever that are commonly referred to malaria and which are of endemic prevalence throughout the continent among all classes of the people, under certain seasonal influences assuming an epidemic character. I indicated the localities in which malaria is most active, and spoke of fevers collectively as being influenced by climatic and seasonal conditions, and I shall now give a description of each type. But first let me make a few remarks on the subject of Indian fevers generally. It has long been my impression that the various forms of Indian fever are closely allied to each other etiologically, and that a combination of climatic and local causes, acting on individuals of certain age, race, and personal susceptibility, accounts for the differences in the fever processes that are set up. The characteristics of intermittent, remittent, or continued fever may be well-marked and distinct, but it is often impossible to draw a line of demarcation between them, merging, as they frequently do, so gradually into each other. Cases occur which, at one or other stage, present the phenomena of all these, concluding with those of enteric fever. Periodicity, which is perhaps the most characteristic feature of malarial fever, is after all but an uncertain guide; for all febrile diseases and non-febrile malarial affections have periods of diminution or exacerbation, more or less definite or regular.

True, there is great difference between a well-marked tertian and remittent or continued fever, but the steps by which the phenomena of one pass into those of the other

render the temperature chart alone an uncertain guide. In this country there may be little difficulty in diagnosing the character of a fever, or in tracing it to its proper origin, but in India there is often great difficulty in differentiating one fever from another, whilst no one symptom or pathological change can be regarded as absolutely pathognomonic of either. It would seem that, the initial process of fever being set up, the course and result is determined by individual peculiarity and by the general nature of the surroundings, rather than by any *one* specific cause. For instance, fevers occur in India which clinically and pathologically resemble specific enteric fever, but they seem to depend on general rather than on specific causes, unless, indeed, it be on one autogenetically produced. Typhus and other specific fevers are not here referred to, but those which owe their origin to a combination of causes and occur not only in Europeans who have recently arrived in the country, but among the whole population. Enteric fever occurs in India as it does in England, and doubtless owes its origin to the same specific cause, for I know of no reason why it should not do so, though I recognise the difficulty of defining it; but as I shall endeavour in my next lecture to show, all fever in India, with diarrhoea, Peyerian ulceration, and typhoid symptoms, is not necessarily caused by a specific contagium derived from faecal matter or from the intestines of another person. Let me say, though, that I regard fevers generally in India as differing from those of temperate climates more in degree than in essential nature, and that the differences are determined chiefly by climate and surroundings. The writings of Pringle, Fergusson, and others show that there is no fever now prevalent in India which has not at one time or other occurred in Europe, nay even in our islands.

The chief factors in the causation of fevers in India seem to be vicissitudes of temperature, meteorological states, terrestrial emanations (malaria) from marshy, damp, or dry soils, or from ground polluted by faecal or other organic matter, or impure water; such also being

determining causes of cholera, dysentery, and diarrhœa. Of their real nature and mode of action we are ignorant, and it is extremely important that they should be thoroughly investigated. Happily we know that they are to a great extent preventable and removable, and that in India attention is alive to the importance of this question. The specific poisons which produce typhus, enteric fever, and some other diseases are probably as active in India and other tropical countries as they are here, but I submit that fever with Peyerian ulceration may and does occur from other causes than fæcal contamination. Nor is there, I venture to believe, any physiological or pathological objection to this ; for disturbed vaso-motor action, whether from malaria or other poison, which in one man produces a congested spleen and stomach, or an enlarged liver, in another congestion and ulceration of the colon, may in a third, under modifying influences, have the effect of congesting and finally ulcerating the small intestine and its glandular structures. This is a question of some etiological importance in regard to preventive hygienic measures.

With reference to the alleged malarial origin of dysentery and cholera, I would remark that they seem closely linked etiologically, and that in some respects they present a closer resemblance to fevers than may at first sight appear, though they do not seem to be always under the same epidemic law of prevalence : witness the comparative statement of cholera and fever in the Madras Presidency during the past seven years.

Annual deaths from Cholera in Madras, and Fever during the Famine and Scarcity Years.

Year	Deaths from Cholera	Deaths from Fevers	Remarks
1874	313	226,220	Shows that fever and cholera are not governed by the same epidemic laws
1875	94,546	252,042	
1876	148,193	230,092	
1877	657,430	469,241	Famine year
1878	47,167	374,443	Effect of famine still operating
1879	13,296	285,477	Ditto
1880	613	209,940	Ditto

Ancient Hindoo authors, observes Mr. Anodochurn Kastogiri, a learned Bengali physician and graduate of Calcutta, mention symptoms of cholera as being prominent in a certain type of fever called jewar-atishar, literally 'fever with excessive diarrhoea,'¹ and he remarks:—'It has been observed that both may break out simultaneously, or one follow in the track of the other. In practice mixed attacks of cholera and fever are frequently seen.' An attack beginning with symptoms of fever may end with cholera, or *vice versâ*. And even recently such an epidemic was devastating Amritsar in the Punjab. A report says:—'Choleraic fever is still raging in Amritsar. Business is at a standstill and nine-tenths of the shops have been closed. A correspondent describes the appearance of the place as a city of the dead. He adds that not a single European has escaped an attack, and that the railway, post, and telegraph officers are working under great difficulties owing to sickness among the *employés*.' And a communication from the medical officers who were present gives details, to which I shall presently refer.

No part of the plains of India is exempt from intermittent fever, though some districts suffer more than others where climatic and local conditions, intensifying the miasmata, cause the fever to assume a severer and remittent type. Remission and intermission, however, are not to be regarded as absolute indications of degree of intensity, for a remittent may be comparatively mild, whilst an ague may become pernicious. The types of malarial fevers are only different modes of expression of the same thing, and such terms as jungle fever, Terai fever, Bengal fever, Deccan fever, Peshawur fever, Mysore fever, Moultan fever, Nagpore fever, Scinde fever, Arracan fever, merely express local varieties with no fundamental differences, though there may be certain subordinate features which distinguish each, depending on the local climatic and meteorological conditions, and which may originate and govern epidemic prevalence, when, under the dominion of seasonal

¹ *Indian Annals*, January 1877.

influences, certain localities are prolific in supplying the cause. Let me give a recent example :—¹

‘The pundits and sirdars of Amritsar, in order to avert fever of a singularly virulent description, which destroys from one hundred and fifty to two hundred daily, and is regarded as another form of cholera, have begun the ceremony known as “Akhand Path,” or continuous unbroken reading of the Grunth, or Bible of the Sikhs. Simultaneously the municipal committee have given orders for the thorough cleansing of the streets; but, as the *Civil and Military Gazette* points out, the labourers will have to be imported, for the sweepers, like almost all the clerks in the public offices, are incapacitated by illness.’

A telegram from Lahore, dated October 4, ran thus :—

‘Two thousand two hundred and sixty-five persons died at Amritsar during the eleven days ending with the 1st inst. One half of these were children. The heavy mortality is almost wholly owing to choleraic fever, which still prevails in many towns in the Punjab. Considering that the Amritsar medical and municipal staff is greatly overworked, and that natives are always reluctant to give information of deaths, the foregoing figures must be below the mark.’

The fever here referred to is described to me by the medical officers who witnessed it—Dr. Duke, of Amritsar, and Dr. Ross, now of Delhi—who have sent me the following information :—

Dr. Duke says :—‘Since my arrival here on November 9, 1881, the fever has offered no special aspect; it has not been of a choleraic type. The symptoms have been those of malarial fever. Our hospital medical wards are filled with pale anæmic men, with dropsy, enlarged spleen, diarrhœa, and dysentery, supervening on fever and starvation; for as the Kashmeeris were attacked with fever, their looms were stopped, and they earned no food. Neither typhus nor typhoid have been observed, nor have any cases of fever with a rash been observed in the city. The

¹ *Allen's Indian Mail*, October 24, 1881.

death-rate has now fallen to 50 per 1,000, only 10 or 15 per 1,000 above the usual state of Amritsar, which has 150,000 inhabitants. I will send you notes on any cases of typhoid or other special fever that may hereafter come under my notice.'

Dr. Ross says:—'My opinion is that the fever was of a special type, which appears in cholera years, and resembles relapsing fever very closely. The course and progress of the disease was distinct from ordinary malarious fevers, and neither typhus nor typhoid existed. Some 12,000 people died in two months or thereabouts; the fever that existed in the jail and civil stations was quite distinct. I send extracts from notes I sent to the Surgeon-General bearing on the epidemic, but have no records of cases.

'The rainfall here in 1881 is reported to have been rather more than twice the average fall of the last fifteen years. It fell principally over the city and around the outskirts of the city. Average rainfall at Amritsar during the past fifteen years was 24·9 inches; the rainfall of 1881 was 52·2. At Tarantaran, a station fifteen miles from Amritsar, 68·5 were registered, this being the maximum rainfall.

'The heavy rainfall which commenced in June caused immense collections of water over a tract of canal-irrigated ground to the north and north-east of the city of Amritsar; the two natural drainage channels, the Guntala Nulla and the city ditch being quite inadequate to carry off the water. The consequence was, that the water-level rose to an unprecedented height, bubbling up like miniature geysers; all the wells became thoroughly polluted, and the water tasted distinctly brackish.

'Fever in the city did not appear in an epidemic form until September; it was preceded by cholera about the beginning of August, of an extremely fatal type, and later on, when masked by fever, there was some difficulty in recognising it in time.

'The fever, which prevailed with its utmost force in

September and the early part of October, appeared to be of the relapsing fever species, but with some affinity to cholera. There were the rigors fearfully severe, headache, insomnia, disordered bowels (often constipated), fever, suppression of urine, with death from coma frequently within a few hours after seizure, but then the rice-water evacuations and vomit of cholera appeared in very many instances during the course of an attack of the fever.

‘The two diseases, cholera and fever, supposing them to be distinct, certainly masked one another so effectually that diagnosis was extremely difficult at times. The people, by the end of October, began to show the exhausting effects of the epidemic fever: enlarged spleen, anæmia, debility, jaundice, and the usual sequelæ, told fatally on their enfeebled constitutions.

‘This specific fever was strictly confined to the city, and to those only who had to go inside on duty.

‘I observed in Kohat in 1869 an outbreak of fever very similar to the Amritsar epidemic, followed by cholera. It was then observed also that it was an impossibility to tell when the cholera commenced, the symptoms of many cases of the fever being so similar. It is not difficult to assign causes for the outbreak. In the first place, Amritsar is fearfully overcrowded. The Kashmiris live like rats in some of the Mohullas, and are just as filthy in their manner of life and selection of food. Secondly, there was a rise of water-level in the wells up to the filth-polluted stratum, the water immediately becoming brackish and impure. Thirdly, the sanitary arrangements (not so very perfect) broke down altogether. Excrement lay in heaps in the lanes and kuchas for days before it could be removed, and even then the manner of removal was obnoxious and ineffectual. The sweepers died in large numbers.’

The reduction of fever mortality since sanitary measures have been introduced into India shows how amenable the causes are to sanitary interference. Martin, writing twenty-six years ago, says: ‘Intermittents, which are most frequent throughout India in the rainy season

attack the European and the native soldier in nearly the same proportion; the mortality in both approximating closely.' Sanitary work has made great strides in India since then, and one result is that the death-rate from fever is three instead of thirteen in the 1,000; it continues higher in the native troops simply because they are not subject to the same sanitary control as the European soldier.¹

Intermittent fever as a consequence of exposure to malarial influence, is frequent, and is much the same in India as in other parts of the world. There are the same premonitory symptoms, chills and rigors followed by pyrexia, diaphoresis, and an apyrexial interval of freedom; the same evidence of disturbed vaso-motor action, congestion and functional derangement of viscera, and the same typical forms—the quotidian, with the recurrence in twenty-four hours; tertian, in forty-eight; and quartan, in seventy-two hours. These have been further divided into double quotidian, double tertian, triple tertian, double quartan, and so on; whilst on the other hand, the intervals have been extended to weeks, months, and years.

But it is needless to trouble you with these details. The average duration of the fit is said to be about sixteen hours in the quotidian, ten in the tertian, and six in the quartan, but these are subject to so much variation that

¹ British troops, Bengal Army, sixteen years, from 1830 to 1845:—

Deaths from all fevers	.	13.25	per 1,000		
"	"	"	1875	2.77	"
"	"	"	1876	2.46	"
"	"	"	1877	2.21	"
					} These three years are normal.

In 1878–79 there was more fever from the famine and Afghan War.

Native Troops.

The Native Army death-rate, twenty years, 1825 to 1844, 18 per 1,000.

1874 = 10.94	}	Normal.
1875 = 13.55		
1876 = 11.29	}	Famine. Dear food.
1877 = 10.90		
1878 = 18.04	}	Dear food. Service in Afghanistan.
1879 = 35.15		

the exact types are exceptional. The period of exacerbation is generally early morning for the quotidian, noon for the tertian, three to five for the quartan.¹ Tertian seems to be most common in Europe, then quartan, and last quotidian. In Africa, the West Indies, and India, the quotidian is most frequent. In Burmah, according to Murchison, 83·5 per cent. were quotidian, and 1·6 per cent. tertian. In India, according to Waring, the observations of several medical officers in various stations throughout India and the Tenasserim provinces show that of 2,574 cases of ague, 1,822 were quotidian, 595 were tertian, 29 quartan, 118 double tertian, 10 irregular; and he further states that of 53,753 admissions of European troops, 51,287 were quotidian with 646 deaths, 2,097 tertians with 12 deaths, and 369 quartan with 2 deaths. Dr. Burton Brown says :—‘At least 95 per cent. of our cases of fever at Lahore are intermittent quotidian agues, about 3 per cent. tertians, and the rest quartan ague, remittent fever *and enteric fever.*’

Morehead says ² :—‘Of 243 cases of intermittent fevers in natives of Bombay, 211 were quotidians, 27 tertians, and of 5 the type has not been recorded. In the European General Hospital the greater prevalence of the quotidian type was noted by him.

Dr. Chevers says :—‘In Lower Bengal intermittent fever assumes the quotidian type in natives, and the tertian in Europeans. In upwards of 27 years’ experience I do not believe that I ever saw tertian in a native. I now and then, but very rarely indeed, had some doubt with regard to the type of a European’s fever. A case of quartan never occurred in my practice; enlarged spleen is

¹ But Waring says that Dr. Geddes, who paid particular attention to this, noted that in the maximum number of quotidians it was from to 2 P.M. to 4 P.M., that of tertians from 9 A.M. to 11 A.M., that of quartans from 12 noon to 2 P.M., and that of double tertians from 9 A.M. to 11 A.M. The large majority occurred between the hours of 9 A.M. and 11 A.M. Waring says, according to his own observations made at Mergui of all types of intermittents, the maximum was at 2 P.M.

² *Researches on Diseases in India*, second edition, p. 17.

extremely common among the native poor of Chittagong and Calcutta. I never saw enlargement of the spleen in a European as a consequence of Chittagong fever. The same remark applies to my Calcutta experience; but when ships used to unload at Mutlah, a swamp 40 miles east of Calcutta, the spleen generally appeared below the ribs in European sailors, within about the first five days of intermittent. A gigantic young North-countryman, otherwise apparently free from all disease, died in my ward of spontaneous rupture of the spleen, within a week from the time of his ship's arrival at Mutlah.'

Quotidian ague is apparently the type in first attacks in India. Tertian occurs more frequently in those who have suffered previously, and in whom alternations of temperature, fatigue, irregularities of living and so on, have re-excited it—being evidence not of recent but of pre-existing disease. According to Morehead, quotidian prevails in the rainy season of the south-west monsoon from May to October. Tertians are met with in the cold season as the result of alternation of temperature, and in those who have resided long in malarious localities, and are frequently complicated with enlarged spleen.

Natives suffer in a greater ratio than Europeans, though there are certain tribes that appear to acquire some immunity. In the European and Native troops of the Madras army in the years 1829 to 1838, there was the following relative prevalence of fevers :—

Class	Aggregate Strength	Admissions with Intermittent Fever	Deaths from Intermittent	Percentage of Admissions to strength	Percentage of Deaths to strength	Percentage of Deaths to Admissions
Europeans .	103·431	13·264	134	13·824	0·129	1·010
Natives .	568 403	95·354	1·381	16·774	0·242	1·448

—WARING.

Showing that 4 per cent. more natives were admitted than Europeans. This is accounted for by the different conditions under which the classes respectively lived. The influence of sex and age is shown in this table :—

Table showing Admissions and Deaths from Intermittent Fever in Madras Army, British troops.

Europeans of Madras Army at large stations for 10 years	Strength of each Class	Admissions Intermittent Fever	Deaths from Intermittent Fever	Percentage Admissions to strength	Percentage Deaths to strength	Percentage Deaths to Admissions
Men . . .	75·121	6·104	57	8·125	0·075	0·950
Officers . . .	2·319	98	0	4·226	—	—
Women . . .	6·559	249	3	3·796	0·45	1·204
Children . . .	9·877	524	8	5·315	0·81	1·526

—WARING.

Men are more liable, two to one, than women of the same class; children suffer at about the same rate, but their mortality is 1 per cent. greater. This is probably due to the fever being complicated with dentition and other affections, or to the difficulty of administering quinine in proper doses to young children (Waring). Officers who are more favourably placed suffered half as much as the men.¹

Dr. Coull Mackenzie, of Calcutta, gives the following statement showing the preponderance of quotidian for the last ten years ending 1881, in two Calcutta Hospitals²:—

¹ In another table of fevers in different localities in India, Waring gives the following results:—

Quotidian	4,458 = 79·7 per cent.
Tertian	900 = 10·1 „
Quartan	102 = 1·8 „
Double Tertians	119 = 2·1 „
Irregular	10 = 0·17 „
	<hr/> 5,589

The proportion of quotidian was larger by 16 per cent. in the Bengal than in the Madras returns, whilst the tertian was about 15 per cent. less.

² The ratio of deaths in each type of intermittent fever is given in a Bengal return thus:—

In 51,289 Quotidian	646 deaths = 1·259 per cent.
„ 2,097 Tertians	12 „ = 0·573 „
„ 369 Quartans	2 „ = 0·542 „

—WARING.

CAMPBELL HOSPITAL.			POLICE HOSPITAL.	
	Admitted	Died	Admitted	Deaths
Quotidian	1,176	88	1,174	Not recorded.
Tertian	78	1	25	
Quartan	12	0	5	
Irregular	149	2	0	
Double Quotidian . .	0	0	0	
Total	1,415	91	1,204	

My own experience confirms the frequency of the quotidian, though it has always appeared to me that the day and hour of the recurrence of the paroxysm are apt to be most irregular and uncertain. This irregularity is perhaps peculiar to Indian paroxysmal fevers, and in temperate climates the periods may be more regular. It is probable, too, that the early use of anti-periodic remedies may modify the natural course of the disease, and, when it does not prevent, may render the return of the paroxysms irregular, or transform ague into remittent—the former showing improvement, the latter, aggravation of the disease. Determinate periods are not of much pathological importance I think: when a man contracts ague the type will depend on himself, his antecedents and his surroundings, not on difference in the nature of the disease.

I need not enter into the various theories of fever which place the *fons et origo mali* either in the blood or in the nervous system, the cause being something inhaled, ingested, or autogenetically produced—which, circulating, affects the nervous system directly through the blood as a carrier, or through the blood itself, altered by the poison. An ague clearly is a neurosis in the outset; the *materies morbi*, acting on the central nervous system, sets up vaso-motor irritation, which causes dilatation and engorgement of the vessels supplied by the splanchnic, the skin and external parts being brought into an opposite condition. The result is the rigor and pallid shrunken skin, whilst there is internal congestion, which is followed

by reaction, when the skin and exterior parts become vascular, the signs of pyrexia appear, followed by profuse sweating and then a return to the normal condition takes place. Such are the phenomena; but why malaria should produce them we can no more say than why strychnia excites or conia depresses the cord, or why one set of nerve-fibres determine contraction, another set dilatation of vessels. I doubt if any right explanation of periodicity in disease will be given until we can explain it in health—until we can give the physiological rationale of the cardiac, respiratory, or catamenial rhythm, of the diurnal pulse and temperature wave, and so on. We can only say that certain conditions are induced by poisons or impressions acting on the centres, which in one case modify, in another altogether derange the normal rhythm, substituting for it altered temperature, tissue-change, and exaggerated neuro-dynamic states as seen in the paroxysms of an ague or in the thermic wave of a remittent fever.

The light thrown on the functions of the nerve-centres during the last quarter of a century enables us to indicate the part disturbed, but we know no more of the intimate nature of the molecular changes which result than we know how quinine diminishes blood pressure or affects periodicity, or how a drop of cobra virus so instantly changes the respiratory centres as to paralyse them; but we do, to a certain extent, know how to modify, control, or even prevent them. The importance of recent investigations into the nature of malaria and the mode of its action cannot be exaggerated, and we owe a debt of gratitude to those who are working in such an earnest spirit to ascertain the truth.

The cold stage of fever is generally preceded by indisposition, lassitude, weariness, muscular pains, dull aching sensations extending along the course of the trunks of the large nerves, yawning, sighing, sneezing, anorexia, thirst, headache, occasionally by a coated tongue, nausea, or vomiting, and sometimes by loose, dark-coloured motions. Slight rises in temperature, sometimes followed by

slight diaphoresis, may, especially in those who have suffered previously, constitute the whole paroxysm, and, passing away, leave the patient well as before. But frequently they usher in a rigor more or less severe—preceding which there is increased elimination of urea (as shown by Professors Parkes and Sidney Ringer) that is continued through the cold and pyrexial stages, diminishing during the intermission until it falls below the standard of health. The amount of urea and the temperature stand in relation to each other, the result of increased metamorphosis, and not of increased elimination after previous retention. The state of the urine of twenty-four hours on a fever day, and on a non-fever day, was examined and compared by Parkes. He found that the watery part is increased before and during the cold stage, and most abundant at the termination of the cold, it decreases slowly during the hot, rapidly during the sweating stage, and does not appear to stand in relation to the quantity of water drunk. The uric acid is also increased considerably during the fit, and after it, urates are deposited freely, though to this there are occasional exceptions. The chloride of sodium is increased during the hot stages (Traube and Ringer) to five times its amount, and phosphoric acid to one-eighth (Nicholson). M. L'Héritier (Waring) gave the density and mean composition in the different stages in twelve patients:—

Specific Gravity	Cold	Hot	Sweating
	1017·330	1020·304	1022·820
Water	967·520	964·680	961·845
Solids	32·480	35·320	38·155
Urea	9·845	9·015	7·624
Uric Acid	0·660	0·980	1·029
Salts and Organic Matter . . .	21·975	25·325	29·502

Zimmermann found the sp. gr. to vary between 1018 and 1025.¹ Dr. Nicholson² found that during the cold and

¹ Casper's *Wochenschrift*, April 1848.

² *Madras Medical Journal*, July 1863.

hot stages the amount of urea was nearly doubled, and the chloride of sodium increased to five times its normal amount. In these stages he noticed a great increase in the quantity of water. In some cases albumen, blood, and renal casts are found (Parkes).

The chilly feeling as if cold water were running down the back soon passes into rigors, the features are shrunk and pallid, the fingers shrivel and turn blue, the skin is rough (goose skin), the body is in a state of tremor, the teeth chatter, and the muscular system is convulsed until the bed or couch is shaken. There is often nausea, sickness, headache. The temperature, notwithstanding the feeling of external cold, is high, and there is a sense of internal heat which is very distressing; this and the nausea or retching are due to the highly congested state of the gastro-intestinal mucous membrane. The temperature begins to rise before the chilly sensation comes on, and attains its maximum, which may be from 105° to 106° towards the end of the hot stage. In absolutely typical cases the temperature returns during the interval to its normal standard. 'This cold stage varies in duration from a mere passing chill with a barely perceptible rigor to severe shiverings, lasting two or more hours; reaction then takes place, bringing in the hot stage. The following is so good an account of an attack of Indian malarial fever, by a layman, that I quote it. The writer is Mr. Edwin Lester Arnold:—

'On the 14th February,' he says, 'it came on, about breakfast time, when a bad headache was rapidly succeeded by a fit of ague, which set me shivering; so that it was scarcely possible to stand up and quite impossible to do any work whilst it lasted, which was about two hours. Then succeeded the hot stage, with sharp pains in every joint and limb, accompanied by a fierce throbbing headache and a terrible thirst which no amount of drink would allay. From twelve o'clock to midnight I was too ill to stand up; then the attack passed off, and by five the next morning, though still weak, I was able to

get to work. (He refers to his work as a coffee planter.) On the top of this, and perhaps resulting from it, I had fever three or four times and began to feel my strength ebbing fast. Generally a sense of oppression about the head, and pains in every limb, heralded the approach of an attack, when I sought the shelter of the hut, and lay shivering or burning all through the long hours of the hot mid-day, while the creepers swung monotonously to and fro and the jungle cicadæ set the forest vibrating with their horrid music. But once I was caught by my enemy when out at work and felt the full force of his power in a way not easily forgotten. I had risen at dawn as usual, and in particularly good spirits accompanied the coolies to the daily purgatory. The sun had scarcely topped the trees when I felt the fever coming on again, the fever increased, my head throbbed and swam, and my teeth began to chatter though there was a burning sky overhead! Still I pushed on, crawling under some logs and over others, but the two miles [to his bungalow] were more than I could manage, and half-way in the middle of a big clearing, my strength ran out, and I sank down on a log, sensible only that, for all the riches which Croesus ever owned, I could not move another step. Then came the cold fit, and the midday sun glared down on me for a couple of hours without for a moment checking the "shivers" which shook me from head to foot. This was again succeeded by the hot stage, when I felt my blood throbbing backwards and forwards like molten lead, and a consuming thirst drove me half mad; but there was not a drop of water to be had anywhere, and not a living thing in sight—nothing but the hot glaring ashes on which I was lying; and the last thing I remembered was sitting up and shouting for water at the top of my voice. I must have fallen asleep after this, for when I roused myself the sun was low down behind the trees; and, limp, weak, and fearfully dirty, I staggered to my feet. Half an hour afterwards a dejected-looking Englishman might have been seen mustering his thick ring of dark-skinned

coolies. How it was got through, goodness only knows. I have an indistinct remembrance of placing a finger on each long native name and reading it over three or four times to get the right sound, while everything swam before me; and, when the end of the column came, suddenly locked myself in, and, just as I was, got supperless to bed.'¹

Dr. Impey (quoted by Waring) found that the average duration of the cold stage in 108 cases was 1 hour and 25 minutes, of the hot 2 hours 3 minutes, and of the sweating 48 minutes. But there were great variations. In 3 cases there was *no* cold stage at all. In two it lasted 10 minutes; in 3 not more than 15 minutes, and in 9 about half an hour. The longest duration of the cold stage was 9 hours. In 240 cases treated by Dr. Waring in Mergui Civil Hospital there was no distinct cold stage in 116 cases. The temperature rises in the body (taken in the axilla and rectum) during the cold stage, but the extremities are below the normal standard, according to Dr. Dunglison,² and Gavarret³ found it so in 6 persons of 18 to 36 years of age—97°·5 to 100°·5—104°·5 F. In five experiments the thermometer stood only 2° higher in the hot than in the cold stage. The cold stage is not always free from danger; when the action of the poison has been very intense, the nervous force seems to be overwhelmed by it; an *algid* state supervenes, the heart fails, the skin becomes cold and clammy and the patient may die in a state of collapse; or, after remaining for hours—it may be forty-eight hours—in this condition, reaction takes place and the hot stage sets in; in other cases, after partial reaction, the symptoms of collapse supervene. Happily this is rare; it so closely resembles the collapse of cholera that if the patient be seen for the first time when in that condition, there might be some doubt as to the diagnosis. Instances are recorded by

¹ *On the Indian Hills*, by Edwin Lester Arnold, pp. 218 *et seq.*

² *Practice of Medicine*, vol. ii. p. 403.

³ *L'Expérience*, vol. x.

MacCulloch and others, of persons having died in a few hours in the Maremma of Tuscany, from the intensity of the poison! Pringle says:¹ 'Several of the men were seized at once with burning heat and a violent headache—some feeling a short and slight chillness before the attack—others mentioning no particular disorder. They also complained of intense thirst, aching of the bones, and pain of the back, great lassitude and inquietude, frequently of nausea, sickness, or a pain about the pit of the stomach, and sometimes they vomited green or yellow bile of an offensive smell. The pulse upon the first attack was generally depressed, but *rose on bleeding*. There were some instances of the head being so suddenly and violently affected that without any previous complaint the men ran about in a wild manner, and were believed to be mad till the solution of the fit by a sweat.' During this stage the spleen and other abdominal viscera and gastric intestinal mucous membrane are much congested; hence the nausea and vomiting that frequently occur. Cardiac action is also depressed and the pulse fails. After a certain duration reaction takes place, the skin becomes hot and flushed, the pulse is quickened, the temperature rises to 103° or 104°, sometimes as high as 105° or 106°, the urine is diminished, and there is thirst; the heart and arteries pulsate violently, the head aches, the temples throb, and delirium may supervene. This stage lasts for an uncertain period, from an hour to twelve or fourteen hours—I saw a case recently in which it had lasted fourteen—but at length gives way; moisture bedews the forehead and gradually the whole body, until the patient sweats so profusely as to saturate the clothing and bedding. This continues for a variable period, when a condition of apyrexia is established and the patient feels relieved, though greatly exhausted; in this state he continues till the recurrence of the next paroxysm which is also not free from danger, for fatal syncope or exhaustion may occur. When this seems to

¹ *Diseases of the Army*, p. 175.

threaten, the patient and his attendants should be warned against an attempt to rise or make any exertion. This has been impressed on me by more than one case. A staff officer in Calcutta had just gone through a paroxysm when I saw him—a long hot stage had passed—he lay pale, exhausted, and bedewed with cold, clammy sweat, but felt much relieved and was reading. He expressed a desire to remove into another room, but observing his depressed condition, feeble voice and pulse, I instructed his attendants on no account to allow him to move; shortly after I left, he rose, made a few steps, sank, and died on the floor. This collapse is most prone to follow a protracted hot stage. After an interval, the paroxysm is renewed; as it approaches, the pulse becomes depressed, slow and weak, and as the cold stage sets in is small and irregular. The phenomena just described may occur only once or twice, but are often repeated, unless broken and modified by antiperiodics or by removal from the place where the fever was contracted. The duration of the stages varies, one or other may even be absent, and though an attack of fever may not exceed a few paroxysms, yet repetitions are frequent and to them various complications are mainly to be attributed. Twining pointed out that: ‘The frequency and obstinacy of visceral diseases which accompany intermittent fever in India are characteristic, and there is hardly any organ which is not sometimes found affected with disordered function or diseased structure in persons who have been long subject to paroxysmal fevers in which there is a frequent return of the cold stage with more or less regularity in its accessions.’ The liver, the spleen, and the portal circulation are prone to be affected, the spleen often very rapidly. But any of the abdominal or thoracic viscera may suffer. These attacks of fever are very liable to occur when the diurnal changes of temperature are great; *i.e.* at the commencement of the cold and during the drying-up seasons. The system, saturated by the poison during the heat and damp, is predisposed to

suffer from the change, and fever is then most readily developed. It is after repeated recurrences of these paroxysms that the visceral changes occur which pass on to a state of chronic disease, producing anæmia, cachexia, and debility. In this condition, and even long before it, the regularity of the paroxysmal returns is broken, and the intermissions are incomplete. Sweating, chills, rise of temperature, general depression of health, disordered secretions, anorexia, pallor, abdominal distension, and diarrhoea supervene, and, if not remedied by treatment or change of climate, may result in hopelessly broken health, or in death; and thus numbers drag on a miserable existence in the more malarial districts, or, if Europeans, seek to regain health by returning to Europe.

This condition of chronic malarial poisoning with occasional returns of ague, neuralgia, brow-ague, hemicrania, or asthma, in an imperfectly developed form, must be familiar to most of my audience; as is also the extraordinary tenacity with which this feverish tendency clings to old Indians who have suffered from intermittent or remittent fever in India, and sometimes too in persons who present little indication of malarial cachexia; though, probably, on examination, some enlargement of the spleen or liver would be found.

The period of incubation varies, depending probably on the intensity of the miasm, the susceptibility of the individual affected, the condition of his health at the time, and so on. Cases have been recorded where the virulence has been so great as to induce immediate collapse; whilst in others it may have been so slight as to cause simply disturbed health or some of the anomalous symptoms known as masked malarial fever; and it is worthy of note that some persons say that after taking quinine for some time the drug itself produces similar symptoms. Those who have suffered before, seem to be most susceptible; indeed, a chill without the intervention of fresh malarial influence is sufficient to develop fever, as is so often seen in cold and damp climates. A few

days in some cases, in others it may be a month, intervene after exposure to malarial influences before a well-marked attack of fever appears, though malaise, headache, &c., may have been present for days. Whether the attack will be ague or remittent depends probably on individual peculiarities, the character of the miasm, and that of the locality. Where a party of men have been exposed to the emanation of some malarial locality, different types of fever may result; one will have ague, another remittent, a third may only feel rather ill, another may have dysentery or even choleraic symptoms. I have known more than one case where a month elapsed after exposure before the first paroxysm of ague ushered in an attack of fever which assumed the remittent form. Simple ague, however, generally occurs earlier, in a few days or even hours. Among the inhabitants of notoriously malarious districts a considerable proportion do not suffer from either ague or remittent, but present a sallow anæmic appearance, with blanched lips and eyelids, pearly eyes, tumid abdomen, weak and irritable heart, hæmic murmurs, and a general appearance of cachexia, dulness, and hebetude; or there may be neuralgia, asthma, albuminuria; or it may be anasarca and ascites. The spleen is enlarged and often indurated, and the liver is also enlarged, but not so frequently as the spleen. The bowels are irritable, diarrhoea is not unfrequent, and as the cachexia progresses there is a tendency in the discharges to assume the white appearance of the so-called tropical diarrhoea. The cachexia becomes more profound, and death follows from asthenia, or, as I believe, not unfrequently, from coagula in the right heart or pulmonary artery.

The appearance of the first paroxysm of ague is not to be regarded as determining the duration of the period of incubation, for symptoms of a less definite kind often precede it, and they at last culminate in the fit; sometimes no distinct febrile paroxysm occurs at all, and the patient gradually lapses into the state of cachexia I have described. Europeans, who generally get away before

matters have advanced so far, are often surprised by having a first attack of ague after leaving. I have repeatedly seen people from Assam and other parts of Bengal, who, during a long residence there, have never had fever, become so affected, the paroxysm probably occurring on board ship, or after arrival in England. In this state, especially where the spleen is enlarged, the patient becomes hæmorrhagic or scorbutic, and there is a tendency to ulceration or gangrene; noma, gangrene of the scrotum, or cancrum oris, frequently occur among natives, and the slightest abrasion or ulceration is apt to pass into a state of phagedæna. Persons who have lived long in India are often subject to inflammatory absorption of the alveolar processes. The gums are spongy and bleed. The bone is absorbed, and the teeth become exposed, and finally lifted up and loose. Inflammation occurring, causes abscess at the root of the teeth. There is a tendency to the formation of fibrinous coagula in the heart and arteries, before referred to; death frequently occurs rapidly with symptoms of apnoea. The tendency to embolism in the arterial circulation is shown in the gangrene of limbs and other parts. I have often seen a limb in peril from the plugging of its main artery. I have elsewhere¹ described the great tendency that there is to this, and how frequently it proves a source of danger and of death in the course of other diseases to persons who have lived for some time in a malarial climate; or operates in a minor degree, producing boils and abscess. And here I would call attention to another morbid susceptibility impressed not only on those who are the subjects of malarial cachexia, but on others who, living in a malarious climate, are subject to its influences. I refer to the so-called urethral fever, so apt to follow on catheterism, however skilfully performed. The mere passage of an instrument will in some cases produce a severe attack of rigors, followed by fever and sweating, and it may give rise to symptoms of a pyæmic nature. Although the subject

¹ *Clinical and Pathological Observations in India*, 1873, p. 95

more concerns our surgical colleagues, I venture to bring it before you, as whatever may result from malarial poisoning must interest all. I have not time to dwell on this point, but have treated of it fully elsewhere, and would ask those to whom the subject may be of interest to refer to what I have written upon it.¹

I may also notice a peculiar form of fever which is apparently connected with a malarial origin, in which the spermatic cords, epididymis, scrotum, and occasionally the prostate gland, are congested and swollen, attended with great suffering, and sometimes accompanied by gastralgia, nausea, and vomiting. The association of this form of fever with elephantiasis is not unfrequently observed, attending the periodic hyperæmia which leads to permanent hypertrophy. This condition is frequently associated with a disordered and dilated state of the lymphatics, and is accompanied by, if not dependent on, periodic returns of fever, which the natives attribute to lunar influences, and by the presence of filariæ in the blood, as pointed out by Lewis, Manson, Bancroft, and others. These febrile recurrences are sometimes called elephantoid fever, and the condition of hypertrophy elephantiasis, which thus seems to be closely related etiologically to other forms of malarial fever.

Professor Verneuil² has called attention to six cases in which glycosuria appeared to be a sequel of malarial influence. In 1859 M. Burdel published facts which seemed to indicate glycosuria as a consequence of malarial fever; he found sugar in the urine of eighty out of eighty-six cases of intermittent fever, but where the fever became remittent the sugar diminished; especially he observed it in pregnant or suckling women and in all cases of pernicious fever. Professor Verneuil concludes that malaria frequently engenders glycosuria in one of two forms—one contemporaneous with the attack of fever, and transient; the other more or less tardy in its

¹ *Clinical and Pathological Observations in India*, 1873, p. 35.

² *Lancet*, p. 1017, December 10, 1881.

onset, independent of the paroxysm, and in all cases permanent—the second probably a consequence of the first. My own experience would confirm the occasional occurrence of glycosuria with or after malarial fever, but whether as a consequence I cannot say; though, in the general perturbation and increased blood pressure that takes place in malarial poisoning, it is probable enough that they may stand as cause and effect.

Pathological Anatomy.

Death in an uncomplicated case of ague is rare. As Maclean says, 'the direct mortality from intermittent fever in India is small. In Bengal, out of a strength of 344,152, with 111,687 admissions, the percentage of deaths to strength was 0·24, of deaths to admissions 0·76.' Death in the remittent and pernicious forms, and from complications and sequelæ, is frequent. The simple attacks involve no change in structure, and indeed, where death occurs early in the pernicious forms, there may be no evident structural change. But malarial influences, when protracted, involve changes in the blood and viscera, especially in the spleen and liver. Those in the blood are secondary, resulting from imperfect elaboration by damaged blood-making organs, destruction of red corpuscles, and transformation of hæmatine into pigment,¹

¹ The following communication from Professor McConnell, of the Medical College Hospital of Calcutta, on pigmentation, is so interesting that I give it in his own words :—

Malarial Pigmentation.

This is a condition I not unfrequently meet with in cases of (1) Remittent Fever; (2) Intermittent Fever, complicated with Enlargement of Spleen and Liver; and (3) in Dysentery, especially the chronic form, and where there is a distinct history of the patient having suffered, at the same time, from malarial fever at intervals; so that I have come to look upon it as quite one of the *post-mortem* signs or evidences of malarial poisoning; in fact, as part of the morbid anatomy of what we call 'malaria.' It is not a *constant* appearance, but still sufficiently common to enable me to say this with some confidence after a very large experience here.

Frerichs and others have long ago pointed out the significance of this pigmentary change, and I would refer to Chap. VIII. vol. i of Frerichs'

high temperature, and in some cases loss of albumen owing to renal disease. The relative proportion of red

' Diseases of the Liver ' (New Sydenham Society's translation, by Murchison), as containing full information on this point. I will only mention here what I have personally observed.

1st. *In the Brain* (cerebrum and cerebellum) the change is very striking, and affects principally the cortical portion of the convolutions. When the hemispheres are incised, in the usual manner, the grey matter of the convolutions presents a *leadен grey* colour, and contrasts remarkably with the white cerebral substance, which is often pale and pearly-white, or may have a dingy tinge. The same appearance is observed in the deeper ganglia (corpora striata, optic thalami, &c.), but the discoloration is not, as a rule, so marked as at the surface. [It must be remembered that this kind of discoloration is quite different to that which is produced in the brain, as in other organs, by *post-mortem decomposition*. I have been very careful to guard against such an error. The specific pigmentation I am describing has been found by me, over and over again, where the autopsy has been performed only a few hours after death, and while all the organs were still fresh and firm.]

I have made frequent microscopic examinations of the condition described, and may relate briefly what I have found : The pigment matter is very dark, granular, and amorphous ; it is seen closely following the outline of the minute capillaries in the cortical or grey substance and filling the minutest of these, having apparently passed through those of larger size. *In no case, however, does the obstruction appear to be complete.** Similar pigment matter is scattered throughout the cerebral substance external to the vessels, and at some distance from them, but in a surprisingly small amount as compared with the dark leaden colour of the grey matter. No other morbid change is observed. In a considerable number of these cases I have also submitted blood taken from the heart (*post mortem*) to microscopic examination, and have found it to contain much dark pigment matter, either free (molecules and granules) or contained within the leucocytes, *i.e.* a general distribution of pigment or *melanæmia*.

2nd. *The Spleen*.—Pigmentation here is, of course, much more frequently

* This is an important point, I think, because Frerichs and others maintain that there is a correspondence between the deposit of this pigment in the brain and certain cerebral manifestations during life (see *loc. cit.* pp. 329–30) ; but, as far as my own observations go, I have never been able to confirm this, the pigmentation of the brain having been found by me quite as deep (well marked) in cases that have died from simple exhaustion or worn out by dysentery, as in cases in which delirium or coma were manifested or were prominent symptoms during life. I look upon the change as parallel to that in the spleen, which we call the *agne cake*—something which, though not so constantly met with as the hypertrophied and blackened spleen, is yet, like the latter, merely a part of the morbid anatomy of malarial diseases, and has no special indications during life. Frerichs is inclined to believe that the obstruction of cerebral capillaries may be complete, and that thus thrombosis and its sequelæ follow.

and white corpuscles is altered, some authorities stating that the latter are increased; there is excess of water, and the red cells are relatively diminished. Hertz (Ziemssen's 'Cyclopædia') says: 'It has not yet been possible to demonstrate a multiplication of the colourless corpuscles.' There is an accumulation of yellowish brown or dark pigment in the blood which is the result of the destruction of the red corpuscles.

This condition of anæmia goes on increasing, inducing dropsy, œdema, and deposit of pigment, which

found, and is to be met with when the other organs of the body remain free, but what I am now referring to specially is the association of such blackened *spleens* with similar changes in the *liver* and *brain* observed in particular cases. The organ, under these circumstances, is usually enlarged, and sometimes considerably so. It is soft and pulpy in consistency, occasionally almost diffuent. The cut surface has a dark olive or dark reddish-brown colour, and the pigment matter may be uniformly or more irregularly distributed. As a rule, it is most plentiful (as seen in microscopic sections)—(a) in the fibrous tissue of the trabecular structure, but is also found (and sometimes profusely) deposited (b) within the cells of the pulp substance around the Malpighian bodies, (c) within small round cells, having the appearance or characters of leucocytes, and (d) *free*, *i.e.* not contained in any cell-structures. It is always very dark, granular, and amorphous.

3rd. *The Liver*.—Here, also, the colour is a dark slate or has an olive tinge (*quite different to that produced by uniform bile-staining*, as, *e.g.*, often in cirrhosis), and the pigmentation is generally *diffuse*. When sections are examined microscopically, the dark, granular, and amorphous colouring matter is found affecting, *i.e.* deposited in, the inter-lobular connective tissue, and by no means confined to this situation (as represented by Frerichs, see his plate, p. 318, *loc. cit.*). I have generally found the hepatic cells also involved, the pigment penetrating the lobules, as it were, and deposited both in the proper secreting cells and external to them (*i.e.* free); in other words, a more or less uniformly distributed pigmentary change. Here, as elsewhere, the colouring matter is dark, amorphous, granular, unaffected by acetic acid, &c.

You will, perhaps, think I have dwelt upon this matter of pigmentation unduly, but it is one I have worked out for myself very largely, and is so little noticed by British authors that I thought you might like to refer to the matter in your lectures. It is an interesting subject from a pathological point of view, though clinically I am unaware of any signs or symptoms by which the change may be predicted, except, perhaps, by repeated examinations of the blood; and, even if predicted, I do not know that we are able by any kind of treatment to check or prevent such pigmentation.

explains the discoloration of certain viscera seen after death, and also the yellowish and brownish colour of the skin in life. This pigmentation has been regarded as of great significance. Hertz says: 'A number of serious disturbances are associated with this condition of the blood, with consequent disturbance of the organs in which it occurs . . . the ashy grey colour of the skin, an interference with secretion of the liver by obstruction of the branches of the portal vein, and of the flow of bile (icterus), secondary atrophy of the liver followed by ascites, hæmorrhage from the stomach or bowels, after obliteration of the numerous hepatic vessels . . . dangerous brain symptoms, delirium, coma, sudden death.' But he further remarks 'that it is true that severe brain symptoms occur where the pigment is absent.' The albumen and fibrine, it is said, are diminished, but I am inclined to believe, as I have elsewhere stated,¹ that the blood becomes hyperinotic, and acquires the dangerous tendency to form clots and emboli.

A careful examination of the blood in this state is much needed. Anthrax and furunculi are attributed to pigmental obstructions of the capillaries, but I would rather attribute them, and other consequences of obstruction, to fibrinosis. The albumen and blood in the urine may depend on passive hyperæmia; but in more chronic cases it probably is a result of chronic renal degeneration.² The mucous membrane of the gastro-intestinal tract in chronic malarial poisoning will be found chronically diseased, the stomach and duodenum being most prone to suffer. Disease may, indeed, occur anywhere throughout the entire tract, and may explain the pathological changes which have been attributed to the specific poison of enteric fever. It may be remarked that the anatomical structure of the lower end of the ileum favours tension during congestion and consequently ulceration.

¹ *Clinical and Pathological Observations in India*, 1873.

² Dr. G. Johnson, F.R.S., tells me he frequently sees cases of albuminuria which he attributes to malaria.

The spleen, during the initial paroxysm, becomes hyperæmic and distended and may be felt below the ribs; it may become hard and friable or soft and pulpy. The pulp is increased and the capsule and trabeculæ are somewhat thickened; it occasionally is so soft as to assume the appearance of a sac full of blood. Inflammatory products may be combined with the hypertrophy (ague cake), and in rare instances the capsule becomes considerably thickened and tough. The spleen sometimes attains a great size, the normal weight of 5 to 7 ounces increasing to pounds, as much as 18 or 20 lbs. having been recorded—even 40 lbs.—whilst it has been found weighing as low as 1 ounce and 19 grains (Russell).¹ In the soft and pulpy state it is easily injured and is frequently the cause of fatal accidents, sometimes spontaneous rupture among natives. As in the liver, a process of contraction of its fibrous elements, a state of cirrhosis, may be induced, the structural change becoming permanent. In this state of the spleen the blood becomes leucocythæmic, and anæmia is the result. Change of climate and treatment may remove the enlargement, but whilst it remains it is an abiding cause of fever and cachexia, and the ague or other manifestations are apt to recur on the receipt of a chill, fatigue, or an attack of indigestion.

The liver also may become chronically congested and indurated and enlarged from interstitial deposit, but not to the same extent as the spleen. It is dark in colour, marked with pigment patches, or may be somewhat softened as well as enlarged. In this condition its functions are impeded, and various complications result. In some forms of remittent there is vomiting and purging, a jaundiced condition of the skin, absence of bile in the dejecta, and deepened colour of the urine. This may be attended with constipation or flatulence, and in some cases with diarrhœa, the food being hurried out of the intestines as a light-coloured feculence. Acute inflammation of the liver seldom occurs in this state, but occasionally it suppurates

¹ *Malaria, its Causes and Effects*, 1880, p 64

insidiously. The rigors that accompany this are liable to be confounded with those of fever, and it is only pain and bulging that at last reveal their real character.

The muscular system may also degenerate, the heart sharing in the change and becoming a source of danger and suffering. I have seen cases of this kind lately in which, while other signs of malarial poisoning had disappeared, the cardiac asthenia remained. Changes also take place in the cerebro-spinal centres and membranes, inducing effusion, exudation, and thickening, causing partial paralysis of the limbs, or obscure cerebral symptoms. There may be also hyperæmia of the cerebral and spinal substance, punctiform extravasation, pigmented patches, discoloration, and effusion of serum into the membranes of the brain or cord, or molecular changes in the centres themselves. Several cases of partial paralysis have appeared before me, and I can recall others in India—one in a distinguished officer to whom it long proved a source of disability. In some of these cases there is but too much reason to fear that the structural changes are of permanent character; still, it is encouraging to know how completely, though it may be slowly, many of these lesions are recovered from, in a temperate climate.

I have alluded to the influence of malarial poisoning on the genital organs and spermatic cords, which become intensely congested and enlarged. The whole area of distribution of the genito-crural nerve and nerves of the cord share in the mischief, whilst the structural changes resulting from exudation or effusion are sometimes serious. The enlarged cords widely distend the abdominal rings and cause hernia, whilst the visceral congestion, extending to the kidneys, gives rise to albuminuria. The kidneys, in some fatal cases of malarial fever, may be found swollen, congested, and undergoing structural changes. In cases that have become complicated with dysentery, which is common enough—indeed, some would say that this is only another form of the same disease—serious structural changes are often found in the colon and rec-

tum; ulceration and thickening in various stages, which are not always confined to the large gut, but extend beyond the ileo-colic valve into the ileum, where ulceration of the mucous membrane and of Peyer's patches resembles that of specific enteric fever.

Effects of Malarial Fever on Wounds.

Mr. Eccles, a surgeon of the Stafford House Ambulances during the late Russo-Turkish campaign, made some observations¹ on certain forms of malarial poisoning and their local effect on wounds, which I can confirm from experience. 'The local effects of malarial fever on wounds differ according to the stages. In the cold stage the discharge decreases in quantity, and sometimes, if it be prolonged, ceases altogether; the surfaces of the wounds will look bloodless or appear congested, and change in colour from a bright red to a dull purple or grey hue—the granulations being pale and bloodless. Pain is rarely complained of, a sense of numbness being often referred to the wound. In the *hot stage* all the local symptoms undergo a decided change—the discharge becomes thick, copious, and sometimes foetid. Sloughing occurs in many cases; the surface of the wound looks angry and inflamed; the granulations are florid, sensitive, and bleed on being touched; sometimes on removal of the dressings the surface of the wound is bathed with blood, the edges are puffy, swollen, glistening and painful, an area of redness extending some distance round the wound, with burning or throbbing in the seat of injury. During the intermission the wound generally resumes its ordinary appearance, but not unfrequently the inflammatory action set up during the hot stage continues, and the local effects remain after the cause has ceased to exist.'

The following cases illustrate some of the conditions I have described :—

¹ *Report of the Stafford House Committee*, p. 152.

CASE I.—QUOTIDIAN.

Medical College Hospital, Calcutta.

M. D. C., East Indian female, aged 35, admitted July 6, 1881. Day of illness uncertain. No enlargement of spleen or liver, no other complication. Bowels constipated; treated with bark and ammonia in mixture. Small doses of ipecacuanha, soda, and cinchona alkaloids twice a day. Aperients. No fever after July 11. Discharged on July 14.

CASE II.—QUOTIDIAN.

Madras General Hospital.

D. P., East Indian male, aged 37, admitted April 22. Had ague since April 19. Rigors begin at about 10 A.M. daily, fever leaves him about 4 P.M. after profuse sweating.

At noon temperature was 103°·8.

24. Ordered quinine, gr. xv.—s.s.

25. Repeated.

26. Quinine, gr. x. bis die.

27. Repeat.

28. Quinine gr. v. ter die.

May 5, discharged cured.

The chart shows the daily recurrence of paroxysms. Temperature 103°·8 on 22nd; 105°·8 on 23rd; 104°·6 on 24th; fell and remained normal after this. (Temperature Chart No. 1.)

CASE III.—QUOTIDIAN.—(DR. WISE.)

Dacca.

Case of quotidian in a young man from a part of the district of Dacca, which had been fever-stricken for several years.

Rám Chunder Dé Bhumika, aged 20, was admitted into hospital on the evening of March 5, 1874, with fever.

His case is of much interest, as he resided, as a domestic servant, for four months in the village of Dásora, in the centre of the fever-stricken tract of Manikgange.

In October 1873 he was attacked with fever. At first he had daily accessions but no diarrhoea. He took English medicines from the dispensary, but they did not benefit him. In November the fever left him, and for nearly a month he was

comparatively well. It then returned, assuming the quartan form; during the last two months he has had irregular attacks.

On March 3, while employed at manufacturing string, he was seized with shivering, aching, great lassitude followed by fever, which lasted several hours and then abated after profuse perspiration. On the 4th he worked as usual, but in the evening the fever returned. At 12 A.M. to-day (5th), preceded by chilliness, great headache and thirst, the fever came back. On admission at 5 P.M., the spleen was found much enlarged, and the tongue was covered with a dirty white fur. Had no vomiting or nausea. Bowels regular; pulse 94; resp. 30; temp. $101^{\circ}7$.

March 6, 7 A.M.—Temp. $97^{\circ}1$; urine sp. gr. 1,010, chlorides abundant. 5.30 P.M., pulse 64; resp. 28; temp. $99^{\circ}7$. One stool to-day; tongue clean, flabby, and indented by teeth; urine 1,018.

March 7, 7 A.M.—Pulse 56; resp. 20; temp. $97^{\circ}6$. Says that he felt feverish last night. Tongue pale and flabby. Liver is enlarged as well as spleen. Urine 1,018. Quinine gr. xxx. ter in die. 5.30 P.M.—Fever began at 4 P.M. Pulse 88; resp. 24; temp. $102^{\circ}3$. Has headache, burning of eyeballs, general pains and thirst. Felt chilly before the accession, and he has passed water frequently since in large quantities. Urine 1,021.

March 8, 7 A.M.—The fever left about 12 P.M. with sweating. The aching of the body, however, is still felt. Pulse 52; resp. 22; temp. $97^{\circ}5$; urine sp. gr. 1,010, chlorides in excess. 5.30 P.M., fever began at 1 P.M. with violent shivering. His body is burning, and every bone and joint is aching; has great thirst but no vomiting. Severe frontal headache, eyeballs painful to pressure; had irritation of bladder during cold stage. Pulse 130; resp. 40; temp. $105^{\circ}3$; urine sp. gr. 1,019, chlorides abundant, no albumen.

March 9, 7 A.M.—The fever did not leave with sweating, but gradually lessened towards 4 A.M. Pulse 76; resp. 22; temp. $90^{\circ}7$; urine 1,015. Castor-oil an ounce at once, and after four hours, quinine twenty grains, 5.30 P.M.—Had three stools to-day. Eyeballs still burning, head heavy; pulse 74; resp. 24; temp. $99^{\circ}6$.

March 10, 7 A.M.—Conjunctivæ yellowish and muddy. Pulse 56; resp. 22; temp. $97^{\circ}1$; urine 1,019. Morning cold and damp. 5.30 P.M.—Only diaphoretics given to-day, sweated slightly at noon. Pulse 70; resp. 24; temp. $98^{\circ}7$; urine 1,020. Milk 4 lbs. daily.

March 11, 7 A.M.—Eyeballs smart and feel hot. Pulse 56; resp. 22; temp. $97^{\circ}1$; urine 1,010, chlorides abundant. Spleen mixture begun to-day. 5.30 P.M.—Two stools to-day. Pulse 64; resp. 24; temp. $98^{\circ}3$.

March 12, 7 A.M.—Two stools during night. Pulse 48; resp. 20; temp. $96^{\circ}4$; urine 1,005. 5.30 P.M.—Pulse 58; resp. 28; temp. $98^{\circ}1$; urine 1,003.

March 13, 7 A.M.—Pulse 46; resp. 20; temp. $96^{\circ}1$; urine 1,016. 5.30 P.M.—Pulse 60; resp. 26; temp. $98^{\circ}3$; urine 1,010, chlorides abundant. Recovered. (Temperature Chart No. 2.)

CASE IV.—QUOTIDIAN.

Medical College Hospital, Calcutta.

A. C., Eurasian, æt. 65, born in India, admitted September 12, 1880. Suffering on admission from ague, pain in the body and limbs, headache, foul tongue, and want of sleep; bowels confined. He appears to have had normal temperature in the mornings, but ague came on in the afternoon and evening. The cinchona alkaloids were administered. He was discharged on October 25. Temperature had been normal for some days.

CASE V.—TERTIAN.

W. A., East Indian, aged 20, admitted July 1, 1881, on the third day of disease. He is a clerk. First attack of ague on June 30; slight enlargement of spleen but no other complication. Treated with diaphoretics and cinchona alkaloids. No fever after July 5. Discharged July 7.

CASE VI.—TERTIAN.

F. D., æt. 23, Eurasian male, admitted April 21, on third day of disease. Is a fairly healthy youth, a compositor. Attacked on April 19, two days prior to admission. There was some bronchitis, but no other complications. Purgatives, diaphoretics, and cinchona alkaloids. No fever after April 24. Discharged on May 1. (Temperature Chart No. 3.)

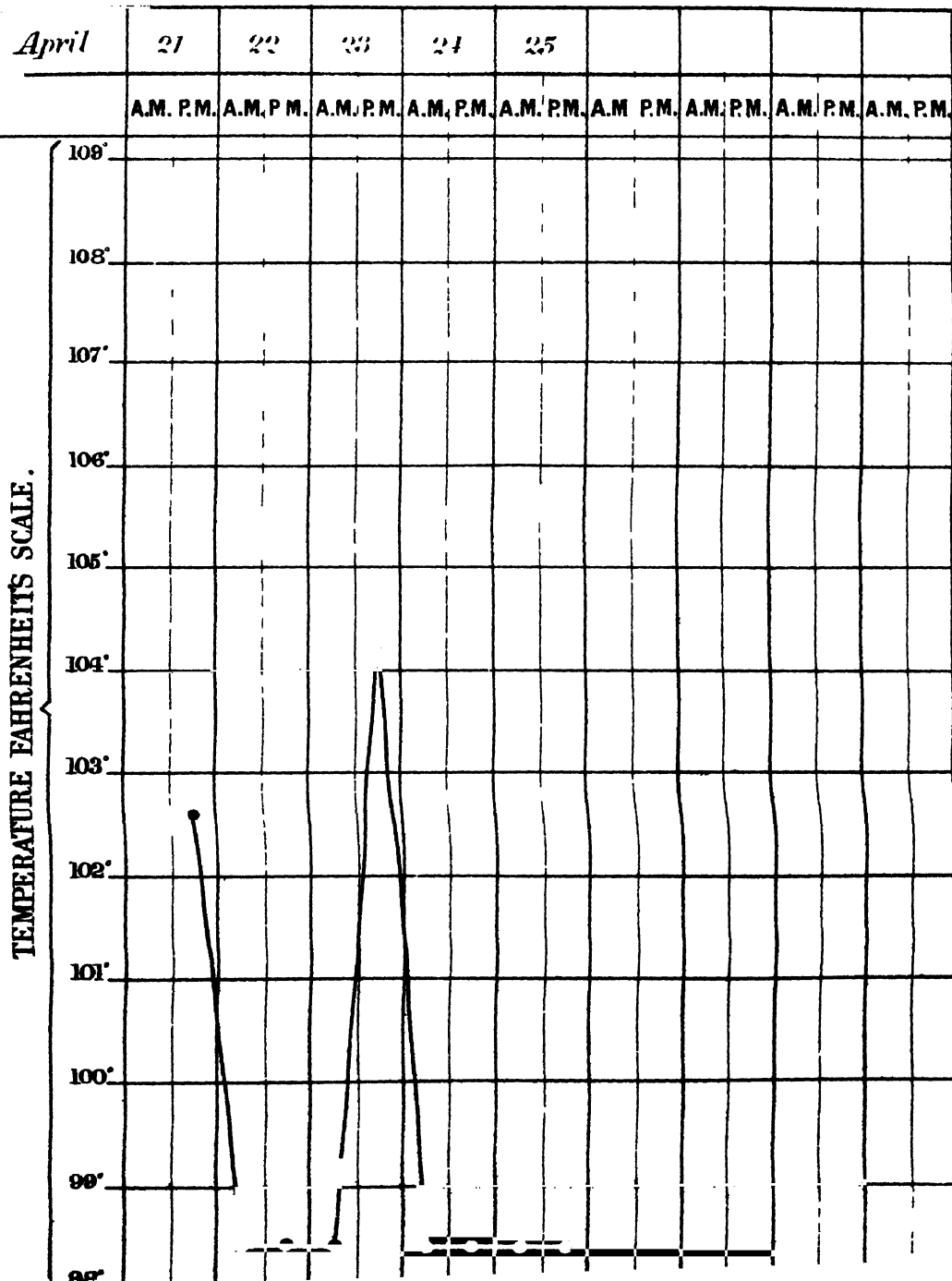
CASE VII.—QUARTAN.

Radha Nath, Hindu male, æt. 27; admitted March 9, 1881, on the 15th day of the disease. He is a cook by trade, well

NAME *F. Dick, (East Indian.)*
Age *23*
Sex *Male*
Place
Disease *Tertian.*
Medical Officer

Case 6.

Result, Cured.



made and nourished. Has suffered from fever for a fortnight; slightly enlarged spleen, and gonorrhœal rheumatism. Treatment: saline diaphoretics and cinchona alkaloids. No fever after March 16. Took iodide of potash for the rheumatic pains. Kept under observation till March 24, when he was discharged relieved. (Temperature Chart No. 4.)

CASE VIII.—QUARTAN.

E. A. C., æt. 38, Eurasian, compositor, admitted January 28, 1881, on the eighteenth day of disease. He says it is eighteen days since the first attack; no hepatic or splenic enlargement. The paroxysm came on the fourth day. Treated with diaphoretics and cinchona alkaloids. No fever after July 30, when he was discharged cured. The influence of treatment is well seen on the chart, the three periodic returns showing gradual abatement in their intensity.

I am indebted to Dr. Jones, of the General Hospital, Calcutta, for the following cases:—

CASE IX.—QUOTIDIAN IRREGULAR.

J. M., æt. 28 years, second mate of a ship; admitted July 29, 1878, with fever. He contracted malarial fever some months ago on the West Coast of Africa. No temperature recorded for the first few days after admission. Suffered from pains in the body and limbs, and ague, with a temperature of $103^{\circ}\cdot4$ in afternoon and night of August 2, which returned on August 3rd, 4th, and 5th; on the 6th, an evening temperature of $102^{\circ}\cdot8$. August 7, free from fever, but in evening temperature of $103^{\circ}\cdot2$, and on the 8th, 9th, 10th, and 11th. Fever continued with but slight intermissions, with ague at times. August 13, morning temperature normal; evening a rise. August 14, hepatic enlargement and tenderness; slight fever, then much better for about a week; then return of ague and fever daily. Checked after six or seven days, after which no return.

CASE X.—IRREGULAR.

T. D. B., aged 24 years; service in India two years ten months. Of nervous temperament.

Landed in Calcutta in good health in November 1878. Three days after arrival was attacked with fever and diarrhœa; improved under treatment, and embarked for Rangoon twi

days later, in a somewhat weakened state of health. Fever returned at sea, and was again accompanied by diarrhoea; the evacuations being at times very frequent and occasionally consisting of blood and slime. Reached Rangoon on November 21, feeling better.

‘Remained in fairly good health for more than a year, and was not under treatment again until February 1880, when suffering from nervous debility.

‘Later on in the same year, after a day’s hard work in swamp and jungle under a hot sun, had an attack of intermittent, the paroxysms occurring every second day. Took 15 grains of quinine daily. Recovery took place in about eight days. From this time until the present attack of fever, which commenced ten days ago, has been in fairly good health. I cannot ascribe the attack of fever to any special cause. It was of an intermittent character, fits occurring on alternate days. Was ordered to take 5 grains of quinine three times a day, and has done so until the date of arrival on July 24.’

Civil Surgeon’s report:—

Mr. B., came under treatment July 24, for ague of the tertian type. During the latter part of last year he was under my care for a similar fever; he was anæmic and emaciated. The paroxysms set in on alternate days, commencing with rigors, succeeded by burning heat of skin, and followed by sweating. During the fever-free days, languor and headache are the prevailing symptoms. There is complete loss of appetite, no power of exertion, disturbed sleep and frequent diaphoresis. Increased doses of quinine were followed by no abatement of fever, but produced more copious sweating. In the course of a few days the character of the fever altered. It was no longer regular, fits of fever became of daily occurrence, and sometimes two or more exacerbations took place on the same day or copious perspiration would set in without any preceding fever. Sometimes an interval completely free from fever occurred, but headache was seldom absent. The quantity of quinine was further increased to 30 grs. daily, given in ten-grain doses at regular intervals.

Later on, 30 and 35 grains were administered in a single dose, but any advance on this produced nausea. In spite of the treatment the fever gained ground, further reducing the patient’s already enfeebled health.

It was obvious that removal was absolutely necessary, and considering that delay might be attended with danger, he was sent to England, where he regained his health, though still subject to occasional slight recurrences of fever.

Remittent Fever.

Remittent fever prevails throughout India and causes much of the mortality ascribed to malaria; it is merely another expression of malarial action, and may commence or end as ague. Intermittents may become irregular, the intermissions being incomplete—some rise of temperature or malaise remaining until the return of the paroxysm. In other cases a period of remitting fever alternates with intervals of complete intermission, and it is not uncommon to hear persons say that they have constant fever for three or four days or a week, which then leaves them, to return in an irregularly intermittent form. These might be called irregular or subintra-intermittents, and they may pass gradually into true remittent, which is a more serious matter, for when it occurs in certain localities and seasons, it is a most dangerous and fatal disease. My first experience of it on the North-East frontier, in natives who had been exposed to the evil influences of the low-lying and intensely malarious country situated between the Cossyah hills and the plains of Sylhet on one side, and Assam on the other, in the valley of the Brahmapootra, taught me its severity, and this from personal experience as well as observation. The cases often rapidly passed into the adynamic condition and were speedily fatal. They were not unfrequently brought in in a semi-conscious condition, soon to pass into complete stupor, with tongue nearly black with sordes, and then death. It is to this form that the term jungle fever, and other local designations, are applied. Of course all jungle fever is not so severe as this, though in certain places and seasons such cases are common. The various local types, no doubt, present some differences in their features. There may be a

more decided icteric tinge, dysentery, diarrhoea, vomiting, choleraic purging, or other indications of gastro-intestinal complication, and these may give rise to such terms as bilious-remittent, malarial dysentery, or choleraic fever. The fever of Peshawur, for example, may present some features that distinguish it from that of Bengal or the Deccan, and so on; but these only point to varying effects of one cause, not to the operation of essentially different causes. The malaria (I use the term conventionally) of an arid district may act differently from that of the jungle or swamp, and, considering the different climatic conditions under which it originates and operates, it is not strange if it do so; but this is a difference of degree only, for the principal features of the fever are identical, and anti-periodic remedies as a rule are efficacious in relieving them. Remittent fever is liable to many complications, and hence has been described as simple and complicated. The period of incubation seems to depend on similar causes to those that determine intensity. Heat, and concentration of malarial poison, may in certain cases act almost immediately; in others, when there is less activity, a few days, to a fortnight or more, may elapse. In my own case it must have been a fortnight or more after exposure in the Assam Terai before fever came on. In the uncomplicated form in a previously healthy person it generally terminates favourably in eight or ten days; but when it is the result of exposure to a more concentrated miasm at the season of greatest activity, or when complicated, it is of longer duration, and may become dangerous and even fatal. In slight cases it is sometimes difficult to say whether it should be called remittent or irregular intermittent (Burton Browne says: 'Remittent fever is generally either a very severe subintrant or more often intermittent fever with some visceral complication'); but in its worst forms it assumes typhoid conditions of the most marked character.

Its access is preceded by languor, malaise, chills, cold feeling in the back and loins, loss of appetite, pain in the

epigastrium, head, back and limbs, vertigo, faintness, nausea, or vomiting, while the tongue is coated with yellowish or white fur, and the pulse is irregular—conditions that may continue for some days before the cold stage (which is not so well pronounced as that of an intermittent, and is sometimes altogether absent) is developed. This gives way to heat, thirst, dryness of skin, severe headache, quick, hard pulse, pain in the eyeballs, and insomnia, the temperature rising to 104° , 106° , 107° ; wandering or delirium may set in and the breathing become oppressed. This state continues for some hours, six or more, often much more, until the remission sets in; the skin then becomes moist, the pulse softer, the pain abates, the sufferer is relieved, perhaps sleeps, though want of sleep is generally a distressing symptom. The remission lasts from six to thirty-six hours or more, when the fever returns as before. The first exacerbation is often the longest, but the second is frequently more severe, and may set in without any premonitory chills or cold stage. If not now checked, succeeding paroxysms may become more severe with scarcely any remission, and a state of great prostration with delirium, unconsciousness, a brown dry tongue, sordes and hiccough supervene—yellowness of the skin and vomiting of bile and altered blood (black vomit) sometimes preceding death. The exacerbation may recur once or twice daily, but there is no regularity; the remission nearly always *takes place in the morning*, and is sometimes so slight that it may be overlooked. The character of the remissions, and the early or deferred return of the exacerbations, are indications of the probable severity or lenity of the attack. Well-marked remissions, free diaphoresis, diminished temperature and headache or cerebral symptoms, are favourable indications; whereas higher fever, ill-defined remissions, accelerated exacerbations, with delirium, coma, and typhoid symptoms, vomiting of blood and bile, or symptoms of collapse appearing as the hot stage is passing away, indicate great danger. The premonitory symptoms are like those preceding

ordinary ague, but the first attack sometimes comes on suddenly, with very little, if any, such warning, and without almost any cold stage before high fever supervenes. But, as far as my experience goes, it generally happens that there *have* been previous attacks of intermittent with imperfect and irregular intermissions; or in very malarious localities the first paroxysm of ague has been succeeded by a remission only. In favourable cases amendment begins in from six to eight days or sooner; the remissions become more complete; the patient sweats freely; the tongue begins to clean and moisten at the edges, and the sordes disappear; the headache and thirst diminish, and the appetite begins to return; he perhaps sleeps and then gradually regains his strength.

Such is the state of things in a simple attack of remittent, and the prognosis is generally favourable; but the brain, the lungs, or the abdominal viscera may become implicated, causing serious complications. Under certain circumstances the symptoms are so sudden and severe as to be mistaken for insolation, and the patient, being overwhelmed at once by the poison, passes rapidly into a state of fatal coma. I have seen such cases in India, and some were recorded by Mr. Eccles, a surgeon of the Stafford House Ambulance, in the Russo-Turkish war, who accompanied a body of men on a long march in which they were exposed to the action of intense malaria and great diurnal vicissitudes of temperature. The type of fever will depend a good deal on the individual who suffers as well as on the circumstances under which he is attacked. In the robust and plethoric young Englishman it will be the sthenic, or, as it has been called erroneously, the inflammatory—the fever high, the pulse full and strong, racking headache, nausea, great thirst, and delirium—which will probably be followed by a well-marked remission and corresponding exhaustion. Such cases are sometimes complicated by exposure to great solar heat, or by abuse of alcohol. In less robust individuals there may be a tendency to the adynamic form; the exacer-

bations reduplicate, the nervous power becomes depressed, and the patient passes into a low tremulous typhoid state, or the fever assumes a continued form. In intermittent fever sudden collapse may occur, and also in this form of remittent; whilst in the sthenic forms cerebral symptoms, suggestive of apoplexy or effusion, may appear. But it is well to bear in mind that such symptoms may be a result of the direct action of the poison on the nerve centres, and that convulsions, especially in children, as pointed out by Dr. Payne, are peculiarly likely to be so caused—such, indeed, being examples of the so-called masked malaria. All acute cerebral symptoms, however, occurring as a result of malarial poisoning, are not of this character; inflammatory cerebral mischief does occur occasionally, and the delirium that passes into coma may be so caused, but the history of the case would encourage hope that the symptoms depended on malarial disturbance. The lungs and bronchi are liable to be implicated, like the abdominal viscera; congestion and inflammation may occur, and natives of India during the cold season are very prone so to suffer. Mr. Partridge, an officer of large experience in Assam, says: ‘I have noticed that after any sudden change in temperature fever becomes very prevalent amongst the (tea) garden coolies. In the rains we may have an interval of hot, dry (dry for Assam) weather, and then a sudden fall of rain accompanied with a cold wind; the sick list is at once doubled or trebled with fevers and pulmonary complications.’ The liver may be gravely complicated and jaundice is no uncommon accompaniment, either as the result of mechanical obstruction of the ducts or of arrested function. Bilious vomiting and epigastric tenderness are of frequent occurrence. The spleen is not so much enlarged, unless there have been previous malarial poisoning, nor is there the same amount of cachexia and anæmia in remittent as in intermittent, unless, indeed, the disease have continued long or recurred often. The kidneys may share in the general abdominal congestion, but albumen in the urine is com-

paratively rare in remittent fever, whilst in the yellow fever of the West Indies it is said to be of constant occurrence. In chronic malarial poisoning, whatever form it may assume, the kidneys may suffer organically, and chronic Bright's disease result. Rheumatism is a frequent complication, especially in the natives; but it occurs also among Europeans, and I not unfrequently find it to be one of the most troublesome symptoms in those who return to England for the recovery of health. It is chronic and affects the extremities and back, but not the heart, unless it have preceded and been independent of the fever. It may be of the ordinary form, complicating the fever, but sometimes it is part of the disease and is another mode in which the effects of malaria are expressed. Dyspepsia and irritability of the stomach are not unfrequent complications of chronic cases, causing debility and cachexia. The usual symptoms, anorexia, flatulence, irregular bowels, irritability, nausea, eructation or vomiting, coated tongue with red edges, occasionally diarrhoea and abdominal tenderness, are present.

Pernicious Forms.

The conditions to which the term 'pernicious' is applied occur either in the intermittent or remittent attacks of malarial fevers as intensifications of any of the stages. The cold stage may be unduly prolonged and occupy the whole paroxysm, the patient either sinking, as in the collapse of cholera, or if he recover, reaction taking place slowly. The hot stage may be intensified and prolonged, the patient becoming delirious, comatose, convulsed, and the condition resembles that of thermic fever, or apoplexy; stupor begins with the commencement of the paroxysms and gradually deepens into complete coma and death, or the symptoms may gradually disappear as the period of the paroxysm passes away. Again, the sweating stage may be very profuse and prolonged, the pulse sinks, and the depression is so great that death

may take place from exhaustion and syncope; or, after the sweating stage has passed, extreme depression may supervene, during which the patient is conscious but hardly sensible of his own weakness, and when any exertion, or even the erect posture, is attended with danger. These are the principal dangers with which I am acquainted. There are many other conditions involving the cerebro-spinal centres and abdominal viscera, of the nature of complications arising out of the general disturbance produced by the malaria rather than by its direct action. I am unable to say what is the immediate cause of the supervention of pernicious symptoms; they sometimes come on suddenly with little warning, after one or two ordinary paroxysms, probably from the intensity of the poison and in subjects who have been unusually debilitated by what has already occurred. It has often seemed to me that in the damp heat of the hot months in certain parts of India, those symptoms which depend on cerebral disturbance are most likely to occur.

Masked Malarial Fevers.

To some of the conditions I have described the term masked fever has been given: neuralgia with imperfectly developed fever, hot hands, aching in the limbs, pain extending along the course of the great nerve trunks in the limbs; gastralgia often causing intense pain and various other forms of disturbed innervation, anæsthesia paresis; functional derangement of the liver and other abdominal viscera, nervous irritability, dyspepsia, asthma, hæmaturia, bronchial irritation, insomnia, and a variety of other symptoms, all pointing to the effects of malarial poison on the nervous system—indeed, all the more severe symptoms that characterise the pernicious attacks have been so termed. The symptoms may be those of collapse, as in cholera, or of apoplexy, epilepsy, cerebral effusion, hæmorrhage from the stomach, bladder, bowel, or kidney, and so on; but the history of the patient and the

circumstances under which the symptoms occur, indicate their true nature, their malarial origin, and their treatment, which generally consists in the use of quinine or some other of the anti-periodic remedies.

These phenomena have generally a periodic tendency, and, like other malarial symptoms, are influenced by the state of the weather, or, as the natives think, at the full or new moon. This subject has been discussed by Indian writers in the 'Medical and Physical Transactions of Bombay,' by Mr. Murray and Dr. Peet. Morehead also refers to it ('Researches on Disease in India'), and says that it is a familiar fact for the hospital physician in India to find several of the asthenic inmates in his wards affected by febrile disease on the same day, though all were free previously, and that these days were coincident with the lunar changes; and that those who have suffered from malarial fever suffered from recurrences on those days. But, as he says—very rightly, I think—as it was generally observed that there were atmospheric changes on those days, the fever was really due to them, not to the moon. Such is probably the real explanation of the so-called sol-lunar influence, advocated by Dr. Balfour, who declared that the meridional periods, diurnal and nocturnal, were distinguished by remarkable changes or paroxysms in the state of the weather, and that these were most remarkable at the lunar periods.'

Malarial Cachexia.

A frequent result of exposure to malarial influences and of repeated attacks of periodic fever is anæmia, and even though there may have been no fever, an enfeebled condition of the health, often a profound state of cachexia, may result, with which is associated structural changes in the abdominal viscera and notably of the spleen. The sufferer has a puffy, blanched face, pearly conjunctivæ and lips, short and hurried respiration, *weak cardiac action, hæmic murmurs, and a feeble pulse;*

a tumid abdomen, not unfrequently dropsy, œdematous lungs and areolar tissue generally, wasted muscles, and a bronzed, discoloured skin, with a large and probably hardened spleen extending down towards, sometimes as far as, the iliac fossa (ague cake). I describe a well-marked case, and such is common enough in the notoriously malarial regions of India, where the whole population presents more or less of this appearance, and where the physical degeneration is accompanied by an almost equally well-marked mental and physical torpor—depression of energy being a characteristic sign of this state. I have known individuals return to this country too late to profit by the change. I have also seen a profound state of malarial anæmia in persons who, having resided for some years in malarial districts, were found to be free from splenic or hepatic enlargement, but in whom the kidneys had suffered, the urine being of a low specific gravity, loaded with albumen and containing renal casts.

The presence of albuminuria is, however, not frequent, nor is it, when in small quantity, of such serious import as might at first sight be supposed. Malarial cachexia may exist with very little obvious visceral complication; though I am inclined to think that if examination be carefully made, some enlargement will generally be found to exist; and so long as it continues, the cachexia will remain and the person be liable to recurrence of fever. Cases of malarial cachexia occur occasionally without fever as the result of the slow but prolonged influence of climate. The process apparently being one of failing health and slowly progressing anæmia, it is indicated by increasing debility, inability for mental or physical exertion, dyspnœa and restlessness, functional derangement of the abdominal viscera, dyspepsia, coated tongue, loss of appetite, chronic rheumatic pains, aching pain along the nerve trunks, neuralgia and other indications of disturbed innervation and nutrition, hot hands and feet, and occasional rises of body temperature. The spleen and liver will occa-

sionally be found enlarged, though it may be but slightly; the urine may be albuminous, the heart's action feeble, and there may be œdema of the feet, face, and probably of the pulmonary tissue. The blood is altered, the red corpuscles are diminished in number and size, and the white corpuscles are relatively if not actually increased. There is a tendency to pigmentation of the skin and tissues, and to the formation of fibrinous coagula, which may prove dangerous if not fatal by plugging the heart or pulmonary arteries, or causing embolism and gangrene. The least wound or abrasion is liable to become phagedænic or gangrenous.

Dr. E. G. Russell, B.M.S., who has resided several years in Assam and paid special attention to malarial diseases and their effects on the spleen, has made some valuable observations on the subject. He says: 'The spleen and stomach suffer directly from malarial poison in a much more marked manner than the liver; the spleen suffers from two forms of enlargement—temporary and permanent; in the former, it suffers in common with the portal system from congestion during the fever, and becomes in fact a diverticulum for the blood of the portal system.'¹ The pulpy substance at the same time increases, so that these temporary enlargements are not mere distensions from excess of blood, but are accompanied with much increase in the amount of the true pulpy tissue. 'The hyperœmia subsiding, this excess of spleen pulp is soon absorbed, it moves on and disappears.' During this temporary enlargement the disintegration and destruction of the red corpuscles is much increased, and hence the exhaustion produced by recurrence of these fevers, which is greater than can be accounted for by high temperature—resembling the effects of severe hæmorrhage; and hence also the anæmia and pallor in cases where the spleen is chronically enlarged. With reference to the recurrence of fever attacks, Dr. Russell

¹ *Malaria, its Causes and Effects*, 1880.

thinks 'the malarial poison is cumulative in its action. It may be and is eliminated as fast as imbibed, and when so no definite ill-results ensue.' The permanent enlargement assumes a chronic form, the congestion becomes combined with a low form of inflammation, and inflammatory products are added. The pulp and trabeculae are found in excess, but the latter not in the same ratio as the former. Such spleens are sometimes abnormally soft, presenting the appearance of a sack of blood; but, on the other hand, the inflammatory products mingled with the pulp may constitute a hard but friable cake easily broken down. As the trabecular tissue does not increase in proportion, the malariously enlarged spleen is seldom met with of tough, firm, consistence. The stomach, duodenum, and liver all suffer from chronic congestion. I would merely add that my own experience of malarial splenic disease entirely supports the views Dr. Russell so ably advocates.

Besides the cachexia and other conditions resulting from enlarged spleen and liver, and a congested portal, renal and gastro-intestinal circulation, there are other sequelae of malarial poisoning which are very distressing, such as neuralgia, which may affect any area of nerve distribution. The fifth nerve, the brachial plexus, and the sciatic are especially prone to suffer; and I have seen many cases in which the suffering was very great. These attacks of malarial neuralgia have their periods of remission and exacerbation, in some instances assuming the quotidian, tertian, or quartan type; and in others being altogether irregular and often very obstinate.

Asthma is also a frequent mode in which malarial poisoning expresses itself, and though frequently purely functional, is liable to produce emphysema and chronic bronchial changes.

The time at my disposal does not permit me to dwell on them at any length, or I should describe the etiological relations of malarial poisoning to elephantiasis, bronchocele, hydrocele, and beri-beri. I have discussed

these questions elsewhere,¹ and must pass on to consider the question of treatment of the malarial diseases I have endeavoured to describe.

Treatment of Malarial Fevers.

The days of bleeding and mercurialism had passed, or nearly so, when I went to India in 1850, so that I have had no personal experience of a mode of treatment which fifty years ago was believed to be absolutely essential. Twining was a keen observer, a thoughtful, highly-instructed, and practical physician, held justly in high repute. His work on diseases in Bengal is still read with profit, for he carefully studied and recorded disease, and has left very accurate pictures of fever as he saw and treated it in Bengal. He says: 'The benefit of bleeding in the cold stage of intermittent fevers is now so well known in India that I hardly need say in a great number of cases it arrests the paroxysm, and is the best mode of preventing those ulterior visceral engagements and indurations which too often prolong the disease till the constitution is ruined.' . . . 'When the cold stage comes on take some blood from the arm at the commencement of the rigors or just where the coldness and shivering are completely established.' . . . 'In general it is sufficient to take 12 or 16 ounces of blood from a European of middle size; in the most robust subjects I would limit the quantity to be taken *at one bleeding* during the cold stage to 20 ounces! In Bengalees I find from 4 to 10 ounces sufficient in general to arrest the paroxysm,' and so on. Again: 'As blood-letting during the cold stage does not always completely remove the existing local affection, it is requisite to ascertain the seat of the predominant inflammation or congestion, and to apply leeches near the part principally affected—from 6 to 10 daily to a plethoric patient in whom general bleeding has been premised; a smaller number every second day

¹ *Clinical and Pathological Observations in India*, 1873.

may be sufficient for those who are emaciated, and after some days' employment of the leeches, it is frequently requisite to apply a blister and keep it open for a week. This follows or accompanies purgatives, and quinine should be administered in the intervals of the paroxysms in the dose of from 2 grains to 4 grains for an adult every two hours. In a few cases I found it necessary to give 4 grains every four hours for four doses when the intensity was so great that a fatal termination was apprehended.' Emetics were not in general use in the treatment of intermittent fever in Bengal, but when there was gastric disorder he administered ipecacuanha in large doses. This was the treatment of intermittent fever in Calcutta in 1830 and for some time after that date!

I have not the statistics for the General Hospital of those days to refer to for the results, but in Twining's ('Diseases of Bengal') records of cases I find that many of them did very well, and he says: 'The practice of bleeding in the commencement of the cold stage has always proved safe and generally more successful than any other remedy.' But he adds: 'There are two sources from which I am apprehensive that Dr. Mackintosh's excellent plan of bleeding in the cold stage may be brought into disrepute—from the operation being trusted to careless assistants, who either do not bleed the patient at the proper moment, viz. the commencement of the rigors, or from a much larger quantity of blood being taken than is requisite to produce a beneficial effect; therefore I have limited the quantity which it is advisable to take at one time.' He says that there are cases in which it may not be desirable to bleed, and he advocates no exclusive practice; neither does he advise bleeding in the cold stage without due consideration of the nature of the disease and condition of the patient. Let me take one illustration¹:—'W. M., æt. 21, tall, rather stout; 9 years in India on July 1, 1830; paroxysm returning every second day at about 11; was freely purged with comp. jalap; on

¹ Twining's *Diseases of Bengal*, vol. ii. p. 220.

July 7, V. S. 14 ounces at commencement of the rigors of the fourth paroxysm, which arrested shivering in less than six minutes. Purgative of colocynth and rhubarb daily. July 13, violent shivering at 7.30 A.M., V. S. 20 ounces! Rigor ceased while the blood was flowing—no hot or sweating stage took place—he had no return of the disease.’ This reads well! rigors ceased and no return. He records other cases of the same character, but not all so speedily successful. Twining is careful to say that he did not bleed his patients for inflammation, but to relieve congested abdominal vessels. But it was a spoliative and needless proceeding, as the practice of later times has shown, though not abandoned altogether I believe in some countries.

I am opposed to all spoliative measures, but I can imagine a condition in which bleeding might be the choice of a lesser evil, and if the abstraction of a little blood could give relief the loss of a few ounces would not signify. Antiphlebotomy, like other things excellent in themselves, may be pushed too far. Needless to say here, that the lancet has been laid aside—calomel emetics and purgatives generally are less used; quinine is more freely given.

How far the mortality of endemic fever in former years may have been attributable to depletion I cannot say, but it certainly has diminished greatly of late years, and death from uncomplicated malarial fever in a European is not frequent now. During an experience of more than twenty years I can remember but few fatal cases, and I think this is the experience of most medical officers who have served in India during the last thirty or forty years.

The statistics of the British Army in India show that the fever mortality from which they have suffered is not due to periodic fevers. Through the kindness of the medical officers of the General Hospital of Calcutta and Madras, and of the Medical College and Campbell Hospital in Calcutta, I have been favoured with notes of cases treated

recently in those institutions. It is interesting to compare them with cases of fifty years ago. The simpler and non-spoliative measures are as successful, if not more so, than those of Twining's time—how much better for the patient afterwards can easily be imagined !

The general plan of treatment of ordinary attacks of intermittent fever in India is as follows:—An aperient, consisting of colocynth and blue pill, to relieve constipation and congestion of the portal system, is generally the first and essential step. This may be followed by some saline aperient, sulphate of magnesia or soda, which it may be expedient to repeat. If there be signs of gastric irritation, coated tongue with red edges, or of a tendency to dysentery or diarrhoea, one or two full doses of ipecacuanha, 15 or 20 grains, may be desirable. During the cold stage, warm drinks, warm clothing, and hot bottles or bricks are useful, and during the pyrexia cooling drinks and ice to the head if there is much pain, together with diaphoretics.

This combination may be given—

R Liquor. Ammon. Acet. ℥ij.
Ether Nitric ℥ij.
Potas. Acet. ℥ij.
Mist. Camph. ℥vj.—M.

One-eighth part every two or three hours.

In the sweating stage, simply rest and quiet are required, care being taken to avoid chills. Quinine should now be given and repeated every three or four hours. I have generally given it in the following form:—

R Quin. Sulph. gr. xl.
Acid. Sul. dil. ℥j.
Tin. Aurantii ℥ij.
Aq. ℥viiij.

M. ft. mist.

One-eighth part for a dose.

The diet should be light; stimulants, unless there be some special necessity, are not required. During the intervals the patient should avoid fatigue, excitement,

or exposure to vicissitudes of weather; and he should continue to take quinine, after the first three or four doses, at longer intervals—say of six hours—until cinchonism begins to appear, when it may be gradually relinquished altogether. Should a second or third attack have occurred, it is well to take a dose about an hour before the onset is expected, and if the first dose of 5 grains have not made a decided impression by postponing or diminishing the paroxysms, 10 grains may be given. Many give 10 grains at first; I have generally found 5 grains sufficient in ordinary cases, and when the time comes for diminishing the quantity the dose may be gradually reduced to 3 grains, and given at longer intervals. The bowels must be kept open, not merely with the object of removing accumulation, but of relieving the portal system, liver and spleen. Quinine will have little effect without this; with it, it is most efficacious in diminishing the intensity, and in many cases of preventing return of fever. In uncomplicated cases of intermittent with no visceral engorgement this mode of treatment will generally be efficacious. Dr. Burton Brown, of Lahore, writes:—‘The cure by large doses of quinine is so certain that we often call it four-day fever. A man gets fever one day, sends for the doctor the next, who gives him a purgative, then 15 or 20 grains of quinine; the third day he feels weak, takes another dose of quinine with some tonic, and on the fourth day returns to his work apparently quite well.’

Persistent return of fever will need larger doses of quinine, or arsenic. Complications, whether cerebral, thoracic, or abdominal, and pernicious forms of fever will require modification of the treatment, to which I shall refer presently.

Treatment of Remittent Fever.

In the treatment of this form of fever antiphlogistic measures are equally inappropriate. The high fever,

racking headache, muscular pains in back and limbs, epigastric pain, nausea, vomiting, and other painful symptoms, call urgently for relief but not by bleeding, unless indeed a few leeches be applied, and that but very seldom. The bowels should be freely acted on by colocynth or jalap, with a moderate dose of calomel, and acetate of ammonia, with ether, to induce diaphoresis; cooling drinks and iced water should be given, whilst ice is applied to the head, which should be shaved if the heat and pain be great; cold affusions, sponging, or even the wet sheet may be used if the temperature is very high; care being taken not to apply cold long enough to cause depression. Draughts of tepid water will soothe the stomach and relieve it of bile and other matters, and if the retching be obstinate, swallowing small pieces of ice is grateful. Effervescing draughts of citrate of potash and iced soda water, and the application of a sinapism or some chloroform on a piece of lint over the stomach will sometimes give relief. Pain over the liver and spleen may be met by hot fomentations; and it may be expedient, if very severe, to apply a few leeches over the painful region; but this is seldom necessary. The most important indication is to watch for any sign of remission, which *generally occurs in the morning*, and is recognised by decrease in the pain, fever, and general suffering, and an appearance of moisture on the skin. A full dose of quinine, 10 to 15 grains in solution, should be administered; 20 or even 30 grains are sometimes given. I doubt if they do more good than 10 grains; but am quite prepared to admit that 15 to 20 grains may occasionally be necessary. It sometimes happens that the stomach will not retain the quinine, and that dose after dose is rejected. If so, it may be given by hypodermic injection or by enema. The former is best, and if the needle is made to enter the subcutaneous tissue *with its aperture turned away from the under surface of the skin*,¹ there is little danger of local mischief, though abscess, sloughing, and erysipelas, and

¹ Mr. Scriven uses a syringe devised by Dr. Buzzard.

even tetanus, have followed the operation. In cases combined with splenic cachexia, it should be avoided if possible, and the precaution should be taken of using a solution of the neutral sulphate, or of borate of quinioidine (of Dr. De Vry), which is very soluble and has an alkaline reaction, dissolving in three parts of cold water. Mr. Scriven, of the Bengal Medical Service, dissolves the sulphate in tartaric acid, and has used it in a number of cases with efficacy and safety in which other modes of administration have failed. He has not found it produce abscess, ulceration, sloughing, or tetanus, if the precautions before mentioned, which were suggested by him, were observed. It is preferable to the solution of the neutral sulphate on account of its higher concentration, the tartaric solution containing one in three, whilst the neutral sulphate is only one in twelve.

Quinine must be continued until the symptoms abate, the remission becomes more perfect, the tongue cleans, and the condition of prostration diminishes. It is not necessary to continue large doses, but enough to keep up a moderate degree of cinchonism, unless the last attack have been very severe. When the fever tends to assume the adynamic form or become continued; when typhoid symptoms appear to be setting in; when there are delirium, sordes of tongue and lips, muttering, tremor of muscles, dry hot skin, abdominal, hepatic, and splenic tenderness with delirium, depressed cardiac action, and feeble pulse; or, on the other hand, when the febrile condition assumes the sthenic type, and no signs of remission appear, or only in the most transient form, quinine should be given irrespective of the remission. I have ceased to regard pyrexia as an obstacle to the administration of quinine, for though this is more effective when given during the remission, I have often seen it of service in adynamic conditions of remittent when given in all the stages; and I have so often known it reduce temperature in other febrile states that I never hesitate to give it during the pyrexia of malarial remittent; while a coated

tongue and confined bowels need not deter in a bad case from employing it. Nourishment and stimulants are to be carefully administered. Pulmonary and bronchial congestion and inflammation are dangerous and frequent complications. Hepatic and bowel complications may also occur, and will require appropriate management. When the adynamic state supervenes, wine, brandy, and other stimulants are necessary, and quinine may be given, combined with decoction of cinchona and ammonia. The amount of alcohol will depend on the state of the pulse, and animal broths, milk, and other similar nutrients will be required. Convalescence must be carefully tended; change of climate should be enjoined as soon as the strength is sufficiently re-established to enable the patient to undertake a voyage.

Quinine is the most valuable prophylactic and therapeutic agent in the treatment of malarial fevers, and it seldom fails if judiciously used. Still, however carefully administered, it will sometimes neither arrest the paroxysms nor alter the character of the fever; indeed in certain cases it seems rather to do harm than good, though as far as my experience goes, such cases are rare. Failure may be due to the state of the abdominal viscera and gastro-intestinal tract. I have heard intelligent natives ask not to have quinine given to them as 'they did not wish to make the fever worse.' But had a dose of calomel and colocynth, or of comp. jalap powder, or some neutral salt been given before the quinine, the effect might have been more satisfactory. The plan of giving the quinine during the sweating stage and intermission is a good one, but the objection to it during the pyrexial stage is groundless, for it reduces temperature and often produces diaphoresis. In ordinary cases a simple diaphoretic is sufficient in the hot stage; but when the remission is imperfect or the tendency to pass into the typhoid condition so often seen in the worst forms of jungle remittent, is threatening, quinine should be given, without reference to temperature, and if there be ever

so slight an appearance of a remission, it is well to seize the opportunity and give a full dose of 10 or 15 grains; in the pernicious forms it is important, as a general rule, to bring the patient rapidly under its influence.

In former days calomel was regarded as an essential part of the treatment, and large and frequent doses were given; an occasional dose of a few grains combined with colocynth may be useful, especially when there are hepatic complications, but beyond this it is not required.

The essential principle of treatment is to keep the bowels open (not purged), relieve visceral engorgement, bring down the temperature, and neutralise the action of the poison. I know of nothing that has such an antipyretic power as the cinchona alkaloids, for they neutralise the poison, diminish blood pressure, decrease the temperature, retard tissue change, and prevent, or modify, periodicity. Their most marked effects are shown in their influence on malarial fevers; but for the reasons I have given quinine is useful in other pyrexial conditions, and I cannot regard its action as a crucial test of the nature of a fever, for it will reduce temperature in continued as well as in paroxysmal fever. Indeed, in the most fervent of all fevers—the ardent or ‘thermic’—when the temperature rises so high as to imperil life, hypodermic injection of quinine has been thought to have a powerful effect in reducing this; and I believe it is now given in enteric fever in this country with this object.

In pernicious attacks, when the symptoms are those of collapse and depression, either in the cold or sweating stages, quinine must be combined with stimulants and warmth. In the cold stage it may sometimes be advantageously combined with opium, whilst warmth is applied to the body generally. When coma supervenes in the hot stage, ice to the head, sinapisms or turpentine stupes to the legs and trunk, and stimulating enemata, in which 30 grains of quinine may be combined, or a few leeches to the mastoid processes, may be useful. Blisters are

sometimes applied. Under the influence of quinine the symptoms may subside, and the fit terminate like ordinary ague. Stertor, congestion, coma with high temperature, seem to suggest active measures, but not those of a depleting character. This masked form of fever may be mistaken for apoplexy or sunstroke; and from the latter there is probably not much difference. If the algid condition appear, warmth, stimulants, sinapisms, and quinine are necessary. In this condition, and especially when there is gastralgia and vomiting, I have found opium afford great relief.

Time does not admit of dwelling on the numerous other antiperiodics; none of them are comparable to cinchona and its alkaloids. The mixed alkaloids now prepared in large quantity in India from cinchona grown in the plantations at Darjeeling and other hill stations have been submitted to trial and have been found very efficacious, though the objection was made that they caused nausea; it seems as though this and other objections are not so serious as at first imagined, and that there is reason to believe that the alkaloids will come into general use. Though economically they have advantages, they will not supersede the sulphate of quinine in therapeutic value.

Quinetum; Cinchona Alkaloids.—Dr. De Vrij brought this preparation to notice, and it is now largely prepared and used in India, and is found to be very efficacious. The nauseating properties perhaps were rather exaggerated. Dr. Verckhuysen says quinetum is of great value as a febrifuge, but that it takes longer to act, and will not replace quinine in pernicious fever. It has the same apyretic effect as quinine, but is less powerful; larger doses are therefore required at longer intervals before the paroxysm than quinine. It produces no unpleasant effects, no noises in the ear, and can be taken by those who cannot take quinine. It is more efficacious in chronic cases, and as a tonic; whilst in masked malaria it is incomparably superior to quinine. My belief is that it is a very valuable drug,

but there is not sufficient evidence to prove that it is better than the sulphate.

Arsenic is a valuable febrifuge and antiperiodic. When quinine does not succeed, arsenic sometimes will do so, given in doses of 5 drops of the liquor arsenicalis. Care must be taken not to continue its use too long, or until symptoms of gastric irritation are set up. In the treatment of the early conditions of malarial fever I have never seen anything to make me think it equal to quinine; but in cases of chronic malarial poisoning, with frequent returns of fever, neuralgia, or other indications of the chronic action of malaria, I have seen great benefit arise from the continued use of arsenic in small doses—3 to 4 drops of the liq. pot. arsenitis twice a day, after food.

Opium, &c.—The antiperiodic powers of opium are probably the chief reason why opium eating and smoking has become so widely spread a habit in China and India. There is little doubt that it does possess such a power, and that in the earlier stages of malarial fever it gives great relief; it relieves pain, soothes, breaks or stops the periodic return of fever, and seems to assist those exposed to malarial influences in resisting them. It has been used for this purpose since the time of Galen. Trotter Lind, and others, last century, prescribed it, and there may be cases wherein it might be expedient to use it now; it would probably be hurtful during the hot stage, yet in the cold and sweating stages it might be beneficial. Waring says that he has seen it cut short the cold stage like a charm, and mitigate the severity of the following hot stage. I have had no experience of it as a febrifuge, and as there are so many others that would better fulfil the purposes required, except in intercurrent conditions, which might complicate malarial fever, I should not resort to it. Many other drugs are spoken of both in the official and native pharmacopœia, but they are inferior to the cinchona alkaloids; viz. arsenic, biberine, salicine, strychnine, atees (aconitum heterophyllum), piperine, ilicin, bonduc nut (fruit of *Cæsalpina bonduchi*), salts of iron, zinc, picric

acid, the mineral acids, and a variety of native drugs, zinc and nitric acid, the hyposulphites and alcohol. These, or some of them, especially iron, may be of service in certain stages of the fever or in the cachexia following it; but in the treatment of the fever quinine is the most effective agent. Atees is much used in native practice, and no doubt is a valuable drug, whether as an anti-periodic, tonic, or as combined with gentian, chiretta, or other vegetable bitters; but it can in no way take the place of quinine, quinetum, or arsenic.

I may not omit to mention the *tincture of Warburg*. I have never seen anything to make me think it better than quinine, though it certainly possesses febrifuge and diaphoretic properties in a remarkable degree. Dr. Maclean and others speak highly of it, and as its composition has been declared, some of the objections to its use have been removed; but I must leave it with this brief notice.

In the treatment of malarial cachexia, with enlarged liver and spleen, the most important steps are change of climate, the judicious use of preparations of iron and quinine, and attention to the state of the portal system—remembering the necessity for relieving portal congestion before we can expect benefit from other remedies. I do not mean depletive measures or excessive purgation, but only gentle action by salines combined with quinine and vegetable bitters; a carefully-regulated and nourishing diet, and protection from all vicissitudes of climate must be enjoined. In such cases benefit may be derived from the saline and ferruginous waters of Germany, and from measures that tend to improve the general health. I need hardly add that a prolonged absence from the country in which the mischief originated is necessary.

In conclusion, I would remark that though the use of mercury is especially to be deprecated in the treatment of disease connected with malariously enlarged spleen, yet the local application of the ointment of the red iodide of mercury, applied as is done in India for goitre, is often most successful in rapidly reducing the size of the spleen;

and that there does not appear to incur much if any risk of mercurialism being induced. Professor Maclean speaks favourably of it, and I can endorse the opinion he has expressed.

One word in regard to a matter which I have omitted. In advanced splenic cachexia the patient should be very careful not to make any exertion; the easily excited heart is provoked at once into over-action, and the result may be rapid dissolution with all the symptoms of pulmonary obstruction. The last instance in which this was forcibly impressed on my attention was in the case of a young Englishman of eighteen or twenty who had returned from India in a profound state of splenic cachexia—the spleen itself descending nearly to the ilium—and with all the symptoms of anæmia present in the most advanced condition. Under the influence of quinine, iron, and good nourishment, he was improving, and there was hope of further progress. One day, in spite of most earnest warnings to the contrary, he got up, walked to the window, and tried to raise or shut it. He got back to bed exhausted and breathless, and died in a few hours.

CASE XI.

The severe action of malaria was observed by Mr. Eccles on the march of some troops from Larissa to Niccala for two days and two nights, over a plain hemmed in by mountains, with two rivers running through it, nearly dry in summer, overflowing in spring and winter, with rich marsh in the centre and tributary streams. The men drank at muddy pools. On only the first night were they exposed to the dews, yet before their destination was reached three men had pernicious remittent; and on reaching Niccala fifty men went into hospital with malarial poisoning, coming on so suddenly that Mr. Eccles thought that they were cases of insolation. Six of them died in the first exacerbation. These he treated as for sunstroke. These cases occurred during the night and day after reaching Niccala. The symptoms were: countenance livid; headache with vertigo in some, while others were comatose; great heat and dryness of skin; temperature, 102° to 106°; full, hard pulse, sometimes rising to 120; dry and

cracked tongue; vomiting; dyspnoea and tumultuous action of heart frequent. The fatal cases sank very rapidly in stupor, and two had convulsions. Noticing in one a slight remission, Mr. Eccles gave twenty grains of quinine and he found this most effectual. All the other cases were unsatisfactory.

CASE XII.

BILIOUS REMITTENT.

Case of remittent, related by the patient, a medical officer.

‘I have not met with many cases of bilious remittent, but I have experienced it in myself; though I fear I can give but a rather confused account of it.

‘I had severe pain in the head, and repeated vomiting of bile, almost pure bile at last, for I could take no nourishment except soda-water, with sometimes a little claret. This almost invariably was rejected directly, soda-water and champagne kept down when I tried it, but this was not until twenty-four hours at least after the attack commenced. Intense pain in the head, almost amounting to torture, of a grinding and cutting character, so intense that I felt inclined to scream, whilst I could not help groaning. Could not rest quietly either in bed or in a chair, wandered in and out of the rooms, and up and down the verandah; did not at first experience pains in my limbs, but afterwards they were of a dull aching nature. Headache was sometimes of this nature, alternating with sharper pain. Bowels costive, with an inclination to be moved, but no result. Sleeplessness or only dozing for a short interval; hot, dry skin, no perspiration, though an intensely hot, muggy, August day.

‘Was detained for some twenty-four or more hours at the rest-house, then returned to Dibroghur, twenty miles, but could not reach head-quarters, and halted half-way. Nausea continued, and pain in the head returned, but no vomiting. Had felt much better when I started in the morning, but rapidly became worse as I proceeded. Was very weak, and, my pony shying slightly I fell off. Could obtain very little sleep that night, and reached my house the next day quite exhausted. To relieve my head, poured two or three jars of water over it, and felt a crash, and something seemed to give way inside, and to the right of the head (effusion of blood into the right internal ear?). Have been deaf in that ear ever since.

‘Tongue was dry and furred; did not note pulse, but both

the carotids and temporals beat strongly. Precordial pain, dyspnoea, no cough. Believe I looked rather livid in countenance, with eyes suffused, &c. Urine very high-coloured, and I remember noticing that it seemed to burn on being passed, and was scanty in quantity. Worse towards and during night, could not sleep, and only got relief by drinking ice-water, and sitting in a lounge-chair under a punka, and being rubbed by my servant. This latter treatment was simply 'heavenly.' I found a few days afterwards that a strong cup of coffee in the middle of the night relieved me much, and I generally got two or three hours' sleep afterwards. This attack continued for at least a fortnight, when I began slowly to mend. Appetite during the attack none; afterwards and for some time very poor and variable. Tried various stimulants, but found none I cared for or that I could take, except madeira, which I enjoyed. Strange to say, that throughout the whole of the attack I was able to smoke, and actually craved for it.

'Previous to the attack, and since then, have suffered with neuralgia of left temple, sometimes very violent in character, which is relieved by free purgation; also had neuralgic pains in the arms. After the attack, and even now, feel very nervous and hypochondriacal, and do not sleep well.'

CASE XIII.

IRREGULAR REMITTENT.

C. V—, an officer, æt. 26, served in India $3\frac{1}{2}$ years. Had fair health for the greater part of the time. He went out shooting in the Canara jungle, and was exposed to the malarious influences of that region after the rainfall in May and June. He appears to have had no decided attack of fever until September 18, and that followed an attack of cholera on September 10; had previously had malaise, and probably slight feverish attacks. Shortly after, he had an attack of fever, for which he was sent home, and the following is the history of his case after his return, and a very typical and instructive one it is. The chief points that it illustrates are: the apparently long incubation period, the irregularity of the periodicity, the effects of change to another climate, the influence of fatigue and exposure in this country in exciting a return of the paroxysm, the extreme irregularity of the symptoms, and the power of quinine on a person not affected with any visceral complications. Beyond tenderness and fulness of

the spleen there was no visceral complication; the heart and lung sounds normal; urine free from albumen, but high-coloured at times. Bowels fairly regular.

Got a chill on November 22 hunting; 23, 24, 25, malaise and slight feverish attacks; had small doses of quinine.

On November 26 temperature rose to $103^{\circ}\cdot5$. Notes do not say if rigor preceded or sweat followed.

27th, temperature $103^{\circ}\cdot5$, morning; temperature $101^{\circ}\cdot5$, evening.

28th, temperature 102° , morning; temperature $102^{\circ}\cdot5$, evening. Intense neuralgia (head) came on; refused quinine. Morphia injected gave relief; profuse sweating.

29th, temperature 102° , morning.

In a few days the fever *abated*. Morning temperature $102^{\circ}\cdot5$, evening temperature 100° . Temperature usually lower in evening, but no regularity in attacks of fever; the *early morning* being the time most free from fever. There were profuse sweats, occurring with the same irregularity.

Nourishment was fairly well taken throughout. Quinine given irregularly, and in different forms, in doses of two or three grains only (frequently less). December 1, in the morning, temperature $100^{\circ}\cdot5$, pulse 78°.

2nd, morning temperature $102^{\circ}\cdot5$, pulse 100.

3rd, morning temperature $101^{\circ}\cdot5$, evening temperature $99^{\circ}\cdot5$.

4th, morning temperature 100° , evening temperature 101° .

5th, morning temperature $101^{\circ}\cdot5$, evening temperature $100^{\circ}\cdot2$.

6th, morning temperature $99^{\circ}\cdot5$, evening temperature 99° .

This marks the end of this attack. He came downstairs. Still had *perspirations*, but no fever. Took small doses of quinine two or three times a day.

Friday, December 9. He was quite free from fever in the morning, but temperature rose to 104° in the afternoon, and from this date high and continuous fever lasted until the 13th. Temperature never below 102° , rising to $103^{\circ}\cdot5$, with profuse night-sweats.

On December 13 there was a remission, but of short duration. Fever came and went for the four following days; he was thus much exhausted.

[It was at this stage that he came under my observation.]

17th, 6 A.M., temperature $102^{\circ}\cdot5$, quinine was given, four grains at 6 A.M., four grains at 9 A.M. Temperature again rose

to 103°·5. At noon five grains quinine given, at 3 P.M. five grains, at 6 P.M. five grains, at 9 P.M. five grains, at midnight five grains.

18th, at 6 A.M. five grains, at 10 A.M. five grains. At 10.30 A.M. of 18th, temperature 99°. Patient completely cinchonised. The quinine was now given less frequently. In the evening, temperature 97°·5.

The profuse sweats continued for two nights longer, and five grains of quinine were given twice a day up to December 23. There have been occasional slight sweats, but the temperature has not exceeded 98°, and has been as low as 96°.

On the 23rd he came downstairs.

On the 29th he went out. The appetite excellent, and return of strength rapid; very little aperient medicine required. He is convalescent and is going to the south of England for change. I saw him on January 4. He was pale, and had occasional perspirations, but no fever, good appetite, and strength much improved. Recommended to continue quinine for ten days.

CASE XIV.

REMITTENT.

Daniel, a native Christian lad, aged 14, was admitted in the Medical College Hospital on July 4, 1881. He states that eight days ago he ate the greater part of a 'jack-fruit.' He suffered for three days after from colic and diarrhoea, no doubt the result of indigestion. Three days ago he was attacked with fever, and since this the stomach irritability has returned, and he is much weakened and depressed.

On admission the skin was hot. The boy very thirsty and restless. The conjunctivæ slightly yellow. Tongue moist but thickly coated. Stomach very irritable; can retain nothing. The vomited matter is bilious. There is tenderness on pressure over the liver, and the spleen is felt enlarged. The bowels are loose. The motions are bilious and slightly slimy. Urine scanty and high-coloured. Has an irritable cough. The chest is clear on percussion everywhere, but dry, rhonchial, and sibilant sounds are audible at both bases, and the expectoration is scanty and frothy. The heart's action is much excited. The pulse is moderately full, soft, and frequent. There are no spots (or eruption of any kind) on the skin.

Was ordered an effervescing saline mixture every three

hours, and a 3-grain powder of *santonin*. The next day (July 5) there was ^{no} improvement. The stools were still frequent (four or five in the twenty-four hours) and bilious, and the stomach was irritable. Two round worms were found in the evacuations from the bowels, and he vomited one. Mixture to be continued.

July 6 (5th day).—Bowels moved six times, but the vomiting has ceased. No more worms. The eyes are congested, and there is a good deal of headache. Cough troublesome. Mixture omitted, and the following prescribed:—

R Ammon. Carb.
Potas. Bicarb. aa. gr. v.
Vini Ipecac. ℥x.
Mist. Cinch. Co. ℥j. Every three hours.

July 7.—Cough easier. A few moist sounds at bases of lungs, with much noisy sibilant breathing, but percussion clear everywhere. Tongue cleaner anteriorly, but still very thickly coated and yellow at the back. The temperature reached 105° this forenoon, and during the afternoon the boy was delirious.

At the period of highest temperature ten grains of neutral quinine were hypodermically injected, and brought down the temperature to 103°·4 in three hours' time, beyond which it did not rise that evening.

July 8 (7th day).—Worse. Delirium continuous. Evacuations are passed into the bed-clothes. Tongue dry. Very restless. Pulse 152, temperature 105°·47.

Mixture omitted and following ordered:—

R Spir. Ammon. Aromat. ℥xx.
Spir. Chloroformi ℥xx.
Spir. Æther. Sulph. ℥xx.
Tinct. Digitalis ℥vij.
Mist. Cinch. Co. ℥j. Every two hours.

Evening.—Bowels only moved once. Still restless and delirious. *Subsultus tendinum*, pulse 148, temperature 104°·8 Fahr. Is fed with difficulty. Quinine, ten grains, again injected hypodermically, and a bromide and *hyoscyamus* draught ordered at 9 P.M. During the night the patient became gradually weaker and weaker, and died semi-comatose at 6 A.M., on July 9.

Post-mortem examination three hours after death. Body

wasted. Rigor mortis strong in the lower extremities, slight in the upper. Pupils a little dilated.

The vessels of the pia mater are a good deal injected, the blood dark. No inflammatory effusion. Only a few drops of pale pinkish serum in the lateral ventricles. Brain substance generally of about normal vascularity and consistency.

The pericardial cavity contains about 3ij of dark-straw coloured fluid. The right cavities of the heart are filled with fluid, dark blood; no coagula. The left chambers are almost empty. Valves and endocardium, &c., healthy.

Lungs.—Anterior margins emphysematous. The bases and posterior margins of both organs are hypostatically congested, and a little deficient in crepitation; rest of pulmonary tissue healthy.

Liver.—Large and heavy. On section *exceedingly soft*, and of a *dark muddy or slate colour, and uniformly pigmented*. Lobular structure indistinct. The large portal and hepatic veins are loaded with dark fluid blood. Gall bladder about three parts full, duct free, and bile thick, of a brownish yellow, and measuring about $\frac{1}{2}$ oz. Weight of liver, 3 lbs. $7\frac{1}{2}$ oz.

Spleen.—Enlarged to about twice its normal size, and proportionately heavy. Capsule thickened, opaque, stretched and tense; substance *exceedingly pulpy and very dark*. The proper structure indistinguishable. Weight, 14 oz.

Kidneys.—Show recent venous congestion; nothing else remarkable.

Stomach.—Mucous membrane thick and corrugated; much injected and vascular throughout. The small intestine is a good deal bile-stained. The glandular (follicular) structures in both small and large intestine are prominent and slightly enlarged, but not much more so than is usual at this period of life. No specific lesions of any kind. The mesenteric glands are a little hyperæmic.

The stomach contains $\frac{1}{2}$ oz. of reddish serous fluid and two live round worms (*lumbrici*); the small intestine about 3 oz. of soft, highly bilious fæculent matter, and also two *lumbrici*; the large intestines about the same quantity of semi-solid fæces.

CASE XV.

REMITTENT.

M. G., an East Indian, aged 19. No occupation. Admitted

into hospital on September 1, 1881. States he has been suffering from fever of a continued type for the last six days.

The patient is weakly and apathetic. Bowels much constipated. Complains of headache and sleeplessness. There is distinct tenderness on pressure over the liver and spleen, but no palpable enlargement of these organs. Skin hot and dry. Tongue furred. No eruption over the body. No respiratory affection. Ordered a dose of castor-oil at once, and 'diaphoretic mixture' 1 oz. every three hours.

September 3 (9th day of fever).—Temperature $98^{\circ}6$. Feels better. Bowels moved four times in preceding twenty-four hours; motions bilious. Complains still of pain over the abdomen. Headache persists. Evening temperature 100° .

September 4.—Was restless and could not sleep last night. Temperature at 9 A.M. to-day 104° ; came down to 102° in the afternoon.

September 7 (13th day).—Temperature $99^{\circ}6$. Less headache; feels better. Tongue moist and cleaner. Bowels acting freely. Ordered one ten-grain dose of cinchona alkaloid. Evening temperature 101° F.

September 8 (14th day).—Complains of cough and frequency of micturition. Except a little dry rhonchus at the back and bases of the lungs nothing abnormal is discovered. The urine is scanty (though frequently passed) and high-coloured (no albumen, &c.).

Ordered:—

R Liq. Ammon. Acet. \mathfrak{z} ij.

Spir. Æther. Nitros. \mathfrak{m} xv.

Potas. Chloratis, gr. x.

Aquæ camph. \mathfrak{z} j. Every three hours.

Morning temperature 105° ; evening temperature $98^{\circ}6$.

September 11 (17th day).—Temperature persistently high. Bowels constipated. Much headache. Pulse full and quick. Tongue a little dry. Temperature $104^{\circ}4$. In the evening temperature 99° , with copious perspiration.

September 15 (21st day).—The temperature still keeps up, and the remissions are noticed to take place pretty regularly now towards the evening, while the exacerbations occur in the morning. Ordered therefore ten grains cinchona alkaloid and two grains calomel. Morning temperature, to-day, 105° ; evening, $99^{\circ}4$. The following day (September 16) the morning temperature

became for the first time normal ($98^{\circ}4$); the tongue looked cleaner and moist; the patient had slept well, and there was but little abdominal tenderness or headache. The calomel was omitted, and two ten-grain doses of alkaloid alone prescribed, to be given at once (10 A.M.), and repeated in an hour's time.

From this date improvement followed, the febrile excitement gradually subsiding, with all its accompanying distress.

From September 17 the temperature remained quite normal. The lad was very weak and reduced, but now soon began to pick up, and was discharged quite convalescent on September 21. (For daily temperature record see Temperature Chart No. 5.)

CASE XVI.

REMITTENT.

Beppin, a Hindu, aged 19. Admitted into the Medical College Hospital on May 19, 1879.

States he has been suffering from strong fever for the last five days; is fairly well nourished; answers questions readily, but is restless, and has an anxious and somewhat 'wild' expression of countenance. The tongue is thickly coated. There is tenderness on pressure over the epigastrium and right hypochondrium, and an inclination to vomit. The spleen can be felt enlarged. The skin is dry and harsh, temperature $104^{\circ}8$. The pulse small, soft, and frequent. Breathing somewhat hurried, but the chest is clear on percussion, and nothing abnormal is heard on auscultation. The bowels are confined. The head was shaved, and cold water applications ordered. A foetid enema was administered, and 10 grains of *neutral* quinine injected hypodermically into the arms.

Evening.—Inclined to be delirious, tries to get out of bed. Has vomited several times (the vomited matter is green and bilious), and can retain neither food nor medicine administered by the mouth. Temperature 103° . Nape of the neck to be blistered, a sinapism placed over the stomach, and 10 grains of quinine again injected hypodermically.

May 23.—The bowels were freely moved (once) during the night. The irritability of the stomach continues. Mind still wandering, but is not so restless. Temperature 100° . Bladder relieved by catheter, as patient has been unable to pass water himself. The urine is bright coloured, acid, loaded with lithates, and contains about one-sixth of albumen. Pulse soft and weak.

Ordered another sinapism to the abdomen, and another quinine injection.

Evening.—No more vomiting, but is quite delirious. Temperature $102^{\circ}4$. The bladder has to be relieved again.

May 24.—Temperature $98^{\circ}6$, pulse small and weak. Continues delirious. Tongue hard and dry; sordes collecting over the gums. Ordered bark and ammonia mixture, and a stimulant (rum).

Evening.—Temperature $101^{\circ}8$, passed water in the bed-clothes. Condition otherwise unaltered.

May 25.—Temperature 100° ; pulse very weak. Highly delirious; is made to swallow nourishment with great difficulty. Died at 1 P.M.

Post-mortem examination $19\frac{1}{2}$ hours after death. Rigor mortis strong in the lower extremities, slight in the upper.

The dura mater is pink-stained. The vessels of the pia mater abnormally injected and full; slight serous effusion into its meshes over the surface of both cerebral hemispheres, but only a small quantity in the ventricles. The brain substance generally a little softened and hyperæmic. Three drachms of dark straw-coloured fluid in the pericardium. The right cavities of the heart occupied by dark fluid blood chiefly (with also small, jelly-like, decolorised coagula). A little dark fluid blood in the left cavities. Valves, &c., healthy. Heart muscle somewhat soft. Both lungs throughout dark and congested, but spongy and crepitant.

The liver weighs 2 lbs. 8 oz. Has a dark purplish colour and is very juicy, much dark fluid blood dripping from the incised surfaces. The lobular structure indistinct. The gall-bladder is full, the bile thick and brownish-yellow, measuring over an ounce.

Spleen enlarged, weighs $11\frac{1}{2}$ oz. Very dark, soft, and pigmented on section. The Malpighian bodies are abnormally large and distinct.

Kidneys show dark venous congestion, but are otherwise healthy. The mucous membrane of the stomach has throughout a dusky red or purplish colour, and is thick, and cedematous at the œsophageal end. The duodenum and upper half of the jejunum are bile-stained. The cæcum, ascending colon and descending colon present a recently injected and vascular appearance, but no follicular irritation or ulceration. The mesenteric glands are normal.

CASE XVII.

REMITTENT.

Ramnarain, a Hindu, aged 20. Admitted October 21, 1880.

Has been suffering from fever, of apparently a remittent type, for the last eleven days, and during this period the bowels have also been very loose.

Body moderately well nourished; conjunctivæ injected. Tongue moist at the edges and coated and dry at the centre. Complaints of pain on pressure over the right hypochondriac region. The spleen is slightly enlarged. Has a troublesome cough, and on auscultation moist râles with wheezing and cooing, &c., are heard in the interscapular space, and at the bases of both lungs posteriorly. Pulse full and quick. Skin hot but moist; temperature 100°.

October 22.—Patient restless and inclined to be delirious. Stools frequent and highly bilious. Cough troublesome, and the expectoration is scanty, frothy, and tenacious.

October 23 to 27.—Becoming quite delirious on the 23rd, the patient remained so more or less continuously. The cough, looseness of the bowels, &c., persisted in spite of treatment. Occasional twitching of the limbs and subsultus were noticed. The tongue dry, and sordes over the gums and teeth.

October 28 to November 2.—No improvement; very restless and delirious. Is fed with difficulty. Stools less frequent but passed unconsciously. High temperature persistent. The patient gradually became weaker, and died exhausted and semi-comatose, the pupils dilating some hours before death.

Treatment.—At first—

R Vini Antimonialis, ℥x.

Tr. Cinch. Co. ʒj.

Decoct. Cinch. ʒj. Every three hours.

Ice applied to the head, and a sinapism to the back of the chest. Afterwards, as the temperature persisted, several hypodermic injections of neutral quinine were given, but seemed to produce but temporary defervescence.

R Mist. Liq. Ammon. Acet. ʒj.

Vini Ipecac. ℥x.

Tr. Aconiti, ℥j, every three hours for more than 24 hours was also given.

admission, weak, tongue furred, bowels regular. August 2, pain at epigastrium, no enlargement of liver or spleen, tongue fairly clean, pulse full and quiet; 3rd to 5th, bowels open, sleeps badly, tongue fairly clean, gurgling in right iliac fossa, no tenderness; 6th to 8th, continuous fever, bowels rather loose, gurgling in right iliac fossa, and cough; 9th to 10th, no looseness, sleeps badly, tongue fairly clean; 10th to 15th, sleeplessness, tongue furred; 16th to 28th, no fever, or but very slight; 28th, fever in the afternoon, after which complete convalescence.

Almost complete defervescence in this case occurred about the twenty-first day, the usual treatment having previously failed to reduce the temperature. There was some looseness of the bowels and gurgling. No spots.

CASE XIX.

CONTINUED REMITTENT.

Under the care of Dr. Joubert.

W. C., æt. 14, ship apprentice, Scotch, one and a half months in India. Admitted August 2, 1880.

Had been suffering from fever for a week before admission. On admission very weak, pulse quick and weak, skin warm, face anxious, tongue moist and coated brown in centre, bowels confined, no pain on pressure of abdomen. The notes of this case are scanty and refer chiefly to the effect of repeated cold baths given from 7th to 12th. The bowels seem to have been confined and the fever to have been very persistent, salicylate of soda tried at first had no effect. The repeated cold baths usually brought down the temperature to normal or thereabouts, but it rose again rapidly. The tongue cleared about the 17th, and remained clean, but the daily evening rise of temperature continued. The chance of a steamer passage home having offered, the boy was sent to sea and got quite well, I heard afterwards, by the time the steamer reached Ceylon. This was one of those cases that a sea trip nearly always cures. (Temperature Chart No. 7.)

CASE XX.

CONTINUED REMITTENT.

Under the care of Dr. Joubert.

J. de L., æt. 18 years, school-boy, East Indian, born in India. Admitted August 24, 1880; discharged September 10. Had been suffering from fever at the Doveton College for seven-

teen or eighteen days before admission. On admission, tongue coated, bowels confined, temperature 101°. The fever persisted, hardly checked, for nine days after admission; the bowels were rather confined, and the tongue furred. No other symptoms appear to have been noted, and the case presented no unusual features.

CASE XXI.

REMITTENT IRREGULAR.

Under the care of Dr. Joubert.

A. C., æt. 32 years, tea-planter (Assam), Scotch, 15 years in India. Admitted March 4, 1880; discharged April 15.

Has been suffering from intermittent fever of eight months, with splenic enlargement. On admission, diarrhœa, spleen somewhat enlarged, cough, loss of flesh, great anæmia, hæmorrhoids, and slight enlargement of liver; tongue clean and pale. Mucous expectoration, loss of appetite. This case looked suspiciously like one of liver abscess on admission; the diarrhœa and cough continuing for some time, with the steady continuance of the temperature above normal. But on the anæmia disappearing, under the influence of diet and iron (reduced) and quinine, the suspicious symptoms disappeared and the patient was fairly well when he went to sea for Australia.

CASE XXII.

REMITTENT IRREGULAR.

Under the care of Dr. Joubert.

J. B., æt. 21 years, tea-planter. Ten months in India, English. Admitted July 27, 1878; discharged September 4.

Was under treatment for quotidian ague up to three weeks before admission, and not well during interval, having diarrhœa and dysenteric symptoms. Had had fever for the past five days. On admission, flushed and nervous; one yellow loose stool, no enlargement of spleen or liver; pain in abdomen. July 28, tongue covered with brown fur and tremulous; one stool only; some gurgling in right iliac fossa. 30th, tongue clean, no stools for two days. August 2, sleeping fairly well, tongue furred and tremulous, throat sore. Bowels confined. 5th, tongue still thickly coated and throat sore, bowels constipated. 8th, much better, tongue quite clean, throat not sore, bowels confined. 11th, convalescent.

This case appears to have convalesced rather suddenly on the 20th day, and may be considered one of that class now usually called 'twenty-one days' fever' in Calcutta. There was looseness of the bowels previous to admission, which would account for the rumbling or gurgling in the iliac fossa noted the day after admission. The bowels were very costive throughout. There were no complications beyond the sore throat. Quinine had no effect on the temperature.

CASE XXIII.

REMITTENT.

Mootasawmey, æt. 19 years, native, a strong young lad, said to be suffering from fever of six days' duration. Admitted July 19, 1879. At first the fever was ushered in by rigors, but since admission it has been of a remittent type, never sinking to the normal temperature; no cause can be assigned. Appetite fairly good. Spleen enlarged two finger-breadths below ribs. Slight mucous râles in chest. He continued in this state, apparently getting no better until August 21, when he showed signs of improvement. His spleen decreased in size, he passed large quantities of urine, and his appetite and strength improved. Discharged October 4, cured.

Treatment.—Quinine, diaphoretics, occasional purgatives, citrate of iron and quinine, and tincture of perchloride of iron. (Temperature Chart No. 8.)

CASE XXIV.

REMITTENT, CALCUTTA.

K. D., æt. 12 years. Admitted March 22, 1878. States that she has had fever for the last five days; commenced one night with shivering, and has not left her since. Fever comes on at irregular intervals. On March 8 a member of the family was attacked with fever and diarrhoea, and died in eight days; another member was attacked on the 12th, and recovered in five days. Is not at all pulled down, talks rationally; tongue coated with white fur, papillæ project through it. No appetite, no headache, no spots on abdomen, no enlargement of liver or spleen; ordered cinchonidine 15 grains at 5 A.M.

March 23.—Temperature fell gradually after a bath. Quinine 10 grains 5 A.M. Santonin 8 gr.

March 26.—Bowels opened five times ; no worms passed. Is bright and cheerful. Slept well. Quinine 15 grains at 4 A.M.

April 2.—Asking for food. Except for daily rise in temperature, appears well. No enlargement of spleen or liver. Motions dark-coloured. Is sweating this morning. Has been taking 15 grains of quinine since March 27 at 6 A.M.

April 5.—Abdomen was tense and tympanitic yesterday ; was ordered oil ; had a copious dark-coloured stool to-day ; going on well.

April 9.—Quinine 10 grains at 12 noon.

April 10.—Stop quinine in early morning.

April 16.—Going on well. Takes her food well. Sleeps well. Tongue clean. Bowels regular ; no enlargement of liver or spleen.

April 18.—Discharged cured.

CASE XXV.

MALARIAL FEVER AND PAROTIDITIS.

Sepoy of 16th N.I., aged 23, was admitted into hospital at Dacca, under Dr. Wise, on April 15, 1873, with remittent fever, assuming a very violent form on 17th. After vomiting he became insensible, and partially recovering consciousness, was found to be hemiplegic on left side.

On May 4, the 20th day of illness, a hard swelling over right parotid gland appeared. On the 10th it opened. By the 27th it had healed.

On June 11, muscular power in limbs was nearly equal, and his mental faculties were unimpaired. Left on this date for nine months' leave.

The above particulars given by Surgeon J. Duke on April 8, 1874.

CASE XXVI.

PAROTID BUBO.

Piari Chand, aged 11 years, was admitted into Mitford Hospital, Dacca, under Dr. Wise, on August 13, 1874.

Her mother states that on July 25 she was attacked by fever. It was attended by constipation, and at night she was delirious. After sixteen days the fever abated, but three days previously, on the 6th, the left cheek began to swell. Since the 10th there has again been fever. This morning the swelling was opened, and about two ounces of thick curdy matter was let out.

CASE XXVII.

Pneumonia followed by parotid bubo and death.

Gogan Mohan Dutt, aged 84, a very cachectic man, was admitted into the Jail Hospital, Dacca, under Dr. Wise, on July 6, 1873, with remittent fever, which was followed by pneumonia. By the 15th the fever had become intermittent, and by the 19th it had ceased altogether. On the 24th he was discharged before he was thoroughly well, and on the 29th he was readmitted with a return of pneumonia. The temperature never rose to 104°, although both lungs were implicated. On August 19 the glands on the right side of neck became swollen and painful. In about a week a large abscess was opened. The fever during the ripening of this abscess had been slight and intermittent in character.

The discharge from the abscess was copious and very foetid. Fever entirely left him on August 29, after which no abnormal rise of temperature was recorded. The drain from this large suppurating mass and his previous bad health completely exhausted him, and he died on September 13.

At the post-mortem examination a sinus was found leading down to the angle of the lower jaw, which was stripped of periosteum and carious. The cavity was filled with very offensive pus. Both lungs were hepatised and almost black in colour. The liver was much enlarged, weighing 3 lbs. 6 ounces. The spleen weighed 9 ounces.

CASE XXVIII.

Bahádur Khan Kabulí, aged 40, was admitted into hospital at Dacca, under Dr. Wise, on August 28, 1874, with parotid bubo on right side. He was pale and cachectic. He stated that about the 14th inst. he was attacked with severe remittent fever and diarrhoea. He got better under treatment, and remained comparatively well for three or four days, when a swelling appeared on the right side, which opened of itself. On coming into hospital a free opening was made, but by this time the abscess had penetrated into the mouth.

CASE XXIX.

MALARIAL COMA.

In August 1880 I saw a gentleman, near London, aged about 70, of powerful physique and presenting little sign of senile

debility. He had lived many years in the West Indies, and had suffered from malarious fever. Since his return to England he had recurrence of malarial fever, and when I saw him it was on the last occasion. He had passed into a state of stupor, but was partially conscious when I saw him; pulse weak, heart's action feeble, temperature not then noted, but it was apparently about normal. He never rallied, and after prolonged stupor of twelve hours became quite unconscious and died quietly.

CASE XXX.

MALARIAL CARDIAC ASTHENIA.

A lady, aged about 34, went to Egypt for the first time in November 1875. Was in perfect health, and remained so through the winter and spring. In June she came to England, returning again to Egypt in November. Remained quite well during this second winter and spring; spent the summer in England, and went out for a third winter. In the month of April was seized with the first attack of fever. It came on with excessive violence, taking the usual form of shivering fit, hot fit, and perspiration, accompanied with very great pain in the back and head. The attacks of fever after this occurred at irregular intervals, five sharp attacks coming in one month.

In May she came home, and at first had no fever, but it returned in full strength in July, and continued for three months so bad that she was often in bed for days. The liver and spleen were congested, sleep became very difficult, and the pain in the back most intense. The usual temperature during an attack was 103° , but it had been as high as $104^{\circ}5$.

By February she had got fairly well, and returned to Egypt, but before long again was seized with fever, though less violently than before. Came back to England for the summer, and again went to Egypt in the winter. A fortnight after arriving there, having been suffering from constant aching and swelling of the feet, she saw the doctor, who after examination said that the heart was seriously weakened, and she was ordered to lie up entirely. After this the heart symptoms got worse, she came to England, and has been very ill since returning to London; at one time suffering much from breathlessness, constant fainting fits, and pain in the heart, which besides being very weak, had become dilated. The heart is now stronger in all ways, general

health greatly improved, and she is able to lead her ordinary life. Treatment :—Arsenic and iron, the latter seldom agreeing, causing diarrhoea.

CASE XXXI.

MALARIAL CARDIAC ASTHENIA.

A now middle-aged military officer landed in India in February 1857, marched up to the Central Provinces, and after some active service and exposure to the sun in the demolition of a fort, was attacked by fever, which continued off and on until he was sent home on sick leave in 1860. After a long period of service in England and Nova Scotia, during which time he enjoyed very fair health, he returned to India in March 1879. At Madras and Bangalore for nearly four months his health was good, but in July he was ordered to Rumpā. Shortly after inspecting a detachment in the Godaverī he found that his health was failing; the least hill brought on violent palpitations of the heart in walking; cantering or trotting when riding also produced the same effect; he appeared before a medical board, and was sent for six months to the coast and Nilgiris. He remained from December 1879 till July 1880 at Ootacamund, but the rarefied air of this elevated region appeared to have a bad effect on the heart, as the palpitations and suffocating feeling continued. Another medical board in July sent him home in 1880, suffering from 'symptoms pointing to dilatation of the heart, probably the result of previous anæmia and debility,' the result of service in the malarial climate of Rumpā. Animal food three times a day, with claret, digitalis, and dialysed iron, soon had a good effect. The heart gradually became stronger, and he was capable of greater exertion than he had been able to take since July 1874. He is endeavouring to arrange an exchange, as he now feels quite capable of serving in a temperate climate, but fears return to India will reproduce the dilatation of his heart. In June 1881 he had improved greatly, cardiac action was normal, no return of fever or of any malarial symptoms. Anæmia has disappeared.

CASE XXXII.

MALARIAL ASTHMA.

A retired medical officer, now in his sixty-fourth year, went to India thirty-four years ago. He was in good health, but had suffered in childhood from moderate attacks of false croup,

spurious or neurotic. Soon after his arrival he served for about three years at Chittagong, where he suffered nearly every month from the moderate but most tenacious intermittent fever for which that spot is noted. He is still liable to attacks at rare intervals. In the twenty-five cold seasons which he spent in India he suffered from spasmodic asthma in twenty-four, the attacks commencing at the onset of his second winter at Chittagong. He generally suffered for about six weeks at the setting-in of any cold weather. There was always considerable bronchitis. The asthmatic attacks were always severe. During the first twelve years he believes that he was on several occasions threatened with death by suffocation, the spasm subsiding just as exhaustive innervation seemed about to end in dissolution. He first returned to England after eighteen years' service. During eighteen months spent at Cheltenham he had a most violent attack of tertian ague, much more severe than any from which he ever suffered in India or elsewhere. He also had a severe attack of bronchitis, *but not the very slightest symptom of spasmodic asthma*. On his return to Bengal he again became subject to asthma. In 1872 he came home and resided for eighteen months in Bayswater, enjoying good health, *and not having a symptom of asthma*, except a vague threatening while staying in a malarious watering-place. The disease recurred on his return to Calcutta. He has now resided in Bayswater for nearly six years, suffering in cold weather from severe attacks of bronchitis, *but never having a sign of asthma*, save a vague sense of chest uneasiness once when on a summer visit to another watering-place, which had measles in its vicinity. He has determined to avoid the seaside in future. He has travelled a great deal by underground railway, and does not at all dread its atmosphere. He does not think a London fog troubles him more than it does his neighbours.

CASE XXXIII.

MALARIAL HEMIPLEGIA.

C. H. B., a medical officer, twenty-eight years of age, two and three-quarters years' service in India; has been suffering from fever and diarrhoea, the result of exposure in Afghanistan, and his health is in consequence much impaired.

His illness commenced at Ali Musjid on August 29, with an

attack of fever, which lasted six days, reducing him in flesh and strength considerably.

On recovery from this attack, he proceeded to Rawul Pindee, arriving there on September 11. On the 15th the fever recurred together with diarrhoea, and both continued with little or no intermission until the end of the month. He then proceeded to Murree, where at first both diseases were aggravated, though he improved again before leaving. After a stay of thirteen days he returned to Rawul Pindee, where the fever again returned, the diarrhoea continuing the same. After a stay of a few days there he went to Lucknow, where he again had fever. The diarrhoea has continued persistently in spite of treatment. He was debilitated and anæmic and had tenderness over the colon, and change to a temperate climate became necessary.

Since the above report, and since passing the board at Lucknow, has been suffering from hemiplegia of the right side with almost complete loss of sensation.

History of the hemiplegic attack: his own account.

‘On April 5, 1880, feeling an attack of fever coming on, I took ten grains of sulphate of quinine. The attack was short but severe, and the quinine also affected me strongly, there being great giddiness, dimness of vision, and ringing in the ears, conditions from which I had never before suffered after taking that drug.

The following day I felt very weak and exhausted, but had no return of fever.

On the 7th again there was no fever, but still a great feeling of exhaustion, and I went to bed very tired and ‘done up.’

After awakening on the morning of April 8, I found that there was complete loss of sensation all over the right side, from the crown of the head downwards, the anæsthesia extending to and being distinctly defined by the mesial line of the body. There was no paralysis of the tongue. This condition continued with little change, except that, whereas at first only sensation was affected, as time went on there was a certain amount of loss of power in the limbs, which, however, may only have arisen from want of use.

On April 18 I started for Bombay, and arrived there on the 21st. On the 22nd I had another severe attack of fever, and

was very much exhausted for a day or two. I then began to feel better, and on April 26 noticed a slight return of sensation in the tips of the fingers, and was able to move both limbs more freely. From that time till I embarked for England, on May 8, I continued to improve, and by the time I had arrived at Southampton I had completely recovered sensation all over the affected side, only a slight feeling of heat and pricking in the palm of the hand and a slight feeling of dulness in the skin over the front of the thigh remaining, both of which have now almost completely disappeared.'

CASE XXXIV.

MELANCHOLIA, DESPONDENCY, AND DELUSIONS FOLLOWING MALARIAL FEVER.

A. S. G., a young officer, aged 25, who had been in India about three years in a malarious district in the Madras Presidency, where from the nature of his duties he had probably been exposed to malarial influences, declined in health, and became the subject of delusions of a religious nature, and great despondency and depression, with inaptitude for duty. He was placed under treatment with bromides, but no improvement taking place he was sent home, the medical officer who saw him last very judiciously remarking :—'He is still in a very depressed state of mind, and deficient memory. He had malarial fever, an important factor to be taken into consideration with regard to his present state.' He came to England accordingly, and after a year's residence has improved. He appears anæmic and weak, but the mental trouble and depression are greatly alleviated, and there is a probability that they will be altogether removed by longer residence in Europe. It is possible that there may have been some hereditary tendency to these conditions, and it is just this that would be intensified and developed into a morbid state, with a liability to periodic recurrence, by malarial poisoning. There is nothing in the patient's physical conformation to suggest any constitutional delicacy. He is a quiet, temperate young man, with no habits that exhaust or injure his nervous system.

CASE XXXV.

MALARIAL FEVER AND RHEUMATISM.

B. H., æt. 37, military warrant officer, served in India over 12 years, in Bengal 8 years. Had several attacks of intermittent

fever during the last year, complicated with rheumatism, which during the rains was sharp and severe. He was sent to England still suffering from frequent irregular recurrences of fever, with rheumatism. The medical officer in January 1880 says:—‘I recommend change to England as essential for the restoration of his health. If he were to remain here, in Bengal, his constitution beginning to be impregnated with malarial poison, he would most likely suffer severely during the coming hot weather.’ In January 1882 a year’s residence in England had quite restored him to health.

CASE XXXVI.

MALARIAL FEVER AND ALBUMINURIA.

L. S., æt. 26, pallid and anæmic. Has been in Southern India six and a half years, coffee planting in the Wynaad. Kept his health well until end of first year, then got malarial fever, of tertian type, which lasted about a fortnight, but afterwards used to come on at quite irregular intervals; never had dysentery or diarrhœa of any consequence or duration. His first station was about 3,200 feet above sea level, that where the fever began was 2,500 feet. It was not till May 1881 that he began to realise that there was anything seriously wrong; he was then becoming breathless and pallid. Arrived in England in June 1881. I saw him in January 1882. He was then better than he had been, though pallid, anæmic, puffy, with œdematous ankles; no fever since he came home, headache occasionally, bowels fairly open, appetite and spirits good.

Hæmic murmurs; heart’s action irritable; pulse quick. Urine sp. gr. 1019. Albumen one-fourth. Spleen and liver not perceptibly enlarged. Warm clothing, milk diet, and a preparation of iron prescribed.

CASE XXXVII.

DEBILITY AND ANÆMIA THE RESULT OF REPEATED ATTACKS OF FEVER.

W. J. B., military officer, æt. 37 years 8 months. Service in India 18 years. Temperate habits, temperament phlegmatic; disease, fever and enlarged liver.

Previous History.—W. J. B. returned to India in October 1874, and for six and a half years enjoyed excellent health, not having spent even a day on the sick list. In May 1879 he suffered from a severe attack of remittent fever, caused by over-

exposure when escorting guns captured at Cabul from Jumrood to Peshawur. Recovery after this attack was very slow, and for some time afterwards he suffered from indigestion, debility, fatigue after slight exercise, depraved secretions, and derangement of all the digestive organs. During the last four months these febrile attacks have recurred, each one being more severe than the last, and the result of those continued attacks is very apparent.

Present Condition.—He has lost flesh considerably. Weight 9 stone 2 lbs., usual weight 10 stone 7 lbs. The repeated attacks of fever have undermined his once excellent constitution, and are becoming more frequent; each successive one leaves him much weaker. His skin has lost its usual clear colour, he is anæmic and somewhat jaundiced. His last attack of fever was very severe. The report on his case says:—‘W. J. B. has been now six years and a half continuously in India. Five years in the Peshawur Valley, together with the hardships to which he was exposed during the Jawakki Expedition of 1877–78, and the Afghan war of 1878–79, have, all combined, told on his constitution. His liver is indurated and enlarged, and digestion impaired. He has lost all his wonted energy, and is not fit to undergo the heat of the ensuing hot weather. I consider a thorough change absolutely necessary to his recovery, and recommend that he may be granted sick leave to Europe. Treatment.—Tonics, quinine, &c., and local applications to epigastrium and liver.’

In January 1882 he had regained his health and returns to duty in India.

CASE XXXVIII.

Anæmia, debility, and cardiac asthenia, with œdema of lower lids; no albuminuria. An example of the effects of long residence and hard work in a malarial climate.

The patient has been treated with quinine, arsenic, tonics, and careful diet, and is much improved.

CASE OF L., BY HIMSELF.

‘I have been in India (Madras Presidency) since my 21st year, 1853. Had two years’ furlough 1870–72, and now since June 1880 (1 year 9 months and 10 days). I have thus had 25 years’ residence in India. In 1859 I was appointed to inspecting duty on the Western Coast (Malabar and Canara), and since then,

till the last few years, I resided *wholly* on that coast, where the climate is very debilitating, and where good nourishing food is not to be obtained. I was sent home in 1870 for general debility, from which I have suffered more or less for the last 15 years, and which has shown itself in chronic dyspepsia, relaxed throat, and elongated uvula, nervous depression, and weakness of the eyes; of late years, too, by torpidity of liver, piles, sluggish action of the heart, and slight chronic rheumatism; hydrocele, from which I began to suffer slightly about the same time, has within the last three years become inconvenient. In November 1879, at Mangalore, had an attack of low malarious fever, which I could not shake off till I got a change of air to the Neilgherries, and then home. I have never since been without some reminder of it, although on the Neilgherries, and on the voyage, and during the first few months of my stay in England, it did not trouble me much. But this year, whilst residing in O., I began to suffer from it again, and had to consult a medical man there. At the same time, the rheumatic pains getting worse, I went at the end of June last to Buxton, for the benefit of the bracing climate and the waters. I got a good deal better there; a slight feverishness at times, and some remains of the rheumatism was all I had to complain of when I left Buxton at the beginning of November last; since then I have been in London, and I have again suffered from slight attacks of fever. A decidedly bad one occurred on Christmas Day, when I had feverishness, shiverings, pains in the joints, and general malaise and prostration, just as I used to suffer in India, although not to the same intensity.

‘I am unwilling to go back almost immediately to the same climate, the same innutritious diet, the same hard work, and to the same places where I suffered from the fever, as I feel I should suffer again in the same way, and not be able to go on with my work. I therefore wish for an extension of leave.

‘I have to state, in conclusion, that my habits are temperate, I do not eat much, and that only of plain and digestible food. My drink is water and cocoa, and at night before going to bed a little hollands and water, but I take nothing else, and at luncheon and dinner only water. I have studied hard and worked hard, and been a good deal exposed in travelling, but in other respects have always had regard to my constitution, which was not originally a very robust one.

‘Perhaps I should mention that one of the symptoms in my case is a pink sediment in the urine.’

CASE XXXIX.

EMBOLISM, THE RESULT OF MALARIAL POISONING.

An English officer, who resided in a malarious district of Bengal, suffered for some weeks from repeated attacks of malarial fever. The fever had left him for some days, giving place to what was thought to be neuralgia of the whole of one leg. He was pallid, anæmic, and emaciated. I found the limb swelled, glazed, and white, like in phlegmasia dolens. It was intensely painful and œdematous, and colder than the other leg. There was no pulsation in the tibials, and the femoral artery was materially diminished in pulsation. This condition had existed for some days and caused intense suffering. It was evidently due to embolism of the iliac, which was interfering with the circulation in the limb, and gangrene was threatening.

The limb was wrapped in cotton and flannel, all stimulating applications were discontinued, and quinine and iron, with a nutritious but non-stimulating diet were ordered. He slowly recovered, and when able to do so, went away for change; but the limb long continued weak.

I made the following remarks at the time:—

‘Had he not been originally a vigorous and healthy young man, free from visceral defects, I believe the limb would have become gangrenous, which might have rapidly proved fatal.

‘The treatment constitutionally in these cases is that of malarial cachexia—quinine, iron, arsenic, good food, a moderate amount of stimulants, and change of air and climate as soon as it can be effected. When gangrene threatens, but has not yet occurred, care should be taken not to precipitate it by overstimulation; where gangrene has taken place, support is needed until the strength warrants surgical interference, which will not be until a line of demarcation has formed.

‘As there is severe pain, opium may be freely given until sleep is obtained or constitutional irritability allayed. When the line of demarcation is fully formed, if the strength have improved, amputation may complete what Nature has begun; and in these cases it will probably be found that no large arteries require ligature, as the main trunks are obliterated and plugged; the numerous smaller branches which keep up the vitality of the limb will have to be tied.’

CASE XL.

GANGRENE FROM MALARIAL INFLUENCES.—CALCUTTA.

Gobindhun, a Mahomedan labourer, æt. 25, admitted June 4, 1865, with gangrene of the right foot and lower two-thirds of the leg. Had malarial fever six weeks previously, which continued for about a fortnight, and was followed by gangrene of the foot; a line of separation formed and was well marked on admission. The leg was removed at its upper third on June 15. Noliatures were required, for the larger vessels were all plugged. He was discharged cured on October 17, the wound having healed.

CASE XLI.

MALARIAL GANGRENE.—CALCUTTA.

Brahmo, a Hindoo female, æt. 30, admitted May 1, 1867, with gangrene of the toes of both feet. Had malarial fever about two months previously, which lasted for five days, and was followed by gangrene commencing at the little toe. Line of demarcation formed. The parts were removed on June 10. The wound was healing when she died of cholera on July 20.

CASE XLII.

PERNICIOUS MALARIAL.—DEATH.

In August 1871 I saw a young and robust Englishman, aged 28, of active, temperate habits, resident for some years in Calcutta, who was reported to be suffering from a swelling in the groin. He was feverish, with pulse 104, having had severe rigors previously. He had great thirst. Bowels acted, voiding greenish matter. It was suspected that he might have hernia. The fever passed off early next morning. I found no signs of hernia. At 2 P.M. next day alarming symptoms came on; at 4 P.M. his breathing was very hurried and his body was covered with cold sweat, with a pulse rapid and feeble. I found him sinking. Dyspnoea intense, breathing gasping and hurried. Face and lips dusky and livid, pulse imperceptible. He was quite conscious. Chloroform inhalation seemed to give some relief, but he died within half an hour.

The post-mortem examination showed that there was no hernia, as diagnosed on first examinations, the inguinal canals being perfectly normal. The glands in the groin were some-

what enlarged. The intestines were normal, and no choleraic fluid was found in them. The lungs were congested and cedematous, and the bronchial mucous membrane was also congested. The heart was normal and firmly contracted; it contained firm decolorised clots extending into both the aorta and pulmonary artery; all else was normal. This appeared to me an example of pernicious malarial action, and of the rapidly fatal effects sometimes produced in the hot, damp malarious season. Death was due to the pulmonary engorgement, accelerated by the fibrinous coagula in the right side of the heart. The overwhelming action of the malarial poisoning accounted for the rapidly fatal result. The patient was a healthy, temperate man, his organs were sound, and he had not been ill for months before this attack. The second paroxysm proved rapidly fatal, despite all attempts to relieve him. Quinine and stimulants were freely given by mouth and injection; warmth and revulsives were also freely applied.

This case occurred at the most trying time of the year, when the temperature was high, probably 90°, and the air saturated with moisture. He had been out and exposed to the sun during the greater part of the day on which he was attacked.

CASE XLIII.

URETHRAL FEVER (MALARIAL).—DEATH FROM FIBRINOUS CONCRETION IN THE RIGHT SIDE OF THE HEART.

W. H., æt. 39, an English sailor, living in Calcutta, a powerful, muscular man, and apparently in perfect health previous to June 18, 1870. On that day he suffered from retention of urine, which he attributed to a slight excess in drinking a day or two before. On the morning of the 19th he applied for relief at the out-patient department, and said he had suffered from slight stricture for the last five or six years, and that on several similar occasions he had had retention of urine. No. 8 catheter was passed without difficulty, and the urine drawn off. He would not remain for treatment of the stricture, although invited to do so. In every other respect he appeared, and said that he was, perfectly well. He was a remarkably fine-looking man, and very intelligent. He returned to the hospital at 3 P.M., saying he felt very ill, and in great but undefined distress about his stomach. He was at once admitted, and the following account was given of his condition since the morning. Soon after the

catheter was passed he had a chill (probably a rigor), and then became feverish, very restless, and so delirious that his friends were obliged to bring him to the hospital. It appears that he had passed some urine tinged with blood after his return home; his bladder was apparently empty on admission. He was feverish and restless, evidently in great distress, complaining of intense thirst and pain across his abdomen or lower part of the chest. There was some tympanites and the tongue was coated. A cathartic enema and hot fomentations to the abdomen appeared to give him relief. Two cathartic pills were ordered at bedtime. On the 20th the pain seemed to have localised itself in the right hypochondriac region, and there was excessive tenderness on pressure over the liver; his breathing was hurried, and his countenance anxious; he still seemed in considerable distress.

He had had several loose but scanty motions during the night; no urine had been passed since admission, but he may have passed some at stool. There was dulness on percussion over the base of the right lung. The breathing was hurried and gasping, but air entered freely into the lungs.

Turpentine fomentations. Effervescing draughts with etherial and ammoniacal stimulants were freely given. Sinapisms were applied over the heart.

The distress rapidly increased, the breathing became more hurried and gasping, and he complained not only of this, but that something was choking him. A stimulating emetic was given which acted slightly. At about 10.30 P.M. he passed water. No improvement, however, took place; repeated stimulants were administered by the mouth and rectum. The difficulty of breathing increased, and he died, as the house surgeon's notes have it, 'in a sudden fit of gasping,' a few minutes before midnight. He was perfectly rational and conscious from the time of admission to the moment of his death, and his struggles for breath were most distressing to witness.

The weather being very hot and damp, the body was examined ten hours after death. The lungs were hypostatically congested, the right most so, and one portion of the middle lobe was solidified and contained a small patch, the size of a pea, like a pyæmic patch. The lower lobe was hepatised. There were one or two very small patches of tubercular deposit in the apices of the lungs; with this exception they were healthy and crepitant throughout. The pericardium contained a small quantity of straw-coloured serum. The heart was normal, but its cavities contained firm, adherent

fibrinous clots. That in the left side extended from the auricle into the ventricle and into the aorta for about three inches. That in the right was larger, and extended from the auricle, through the ventricle, on into the finer ramifications of the pulmonary artery. The pleuræ were normal. The liver was rather large, but apparently otherwise normal. There was no sign of inflammation in its substance or on its surface. The spleen was normal. The kidneys were congested and large, their capsules easily separated, and granular degeneration was apparently commencing. The other viscera and the peritoneum were perfectly healthy. The bladder was somewhat thickened, and the urethra was slightly strictured in front of the bulb. There was no wound, but it was a little congested where it had bled after the catheter. There was no false passage. The prostate was natural, as were the tissues about the neck of the bladder.

Remarks.—This is a very striking and interesting as well as instructive case. A man in the prime of life, in good health, with the exception of a slight stricture, which was only troublesome when irritated into spasm by such irregularities as that of taking a little more beer than usual; a steady, intelligent, and otherwise temperate person, suffers from retention of urine, due to a slight excess a day or two previously. He applies for relief at the hospital, and is relieved at once by the passage of a No. 8 catheter. He returns home, feels chilly, has rigors, rapidly followed by fever and delirium; he passes urine tinged with blood after his return home. The fever is attended with intense restlessness and distress; severe pain in the right hypochondriac region and pit of the stomach follows. Rapid, deep, and gasping breathing, with the greatest præcordial distress increase, and go on getting worse and worse, until the patient dies in great agony of breathlessness about forty hours after the catheter had been passed. His intellect was perfect to the last; urine was secreted and voided not long before death. It is clear, therefore, that the symptoms were neither due to uræmic nor cholæmic poisoning, nor to any cerebral disorder.

There was nothing to point to cholera or other exhaustive disease as the cause of death. Air entered the lungs freely, and his voice was natural to the last. He did not die of asphyxia. The sounds of the heart were normal, and heart disease was not present. There was no evidence of either peritonitis or other acute inflammation. What then was the cause of death?

The post-mortem examination revealed a congested state of the base of each lung with consolidation of a small portion, and a patch of broken-down tissue about the size of a pea. The pleuræ were healthy, and the pericardium contained a small quantity of fluid; but these conditions were not sufficient to cause death. The abdominal viscera, it is true, were not absolutely healthy, for the liver was slightly enlarged, and the kidneys were congested and in an incipient state of degeneration. The bladder, the prostate, and the tissues about its neck were generally healthy, though the bladder was somewhat thickened, in consequence of a slight stricture situated just in front of the bulb of the urethra. Through this an instrument had been passed. There was nothing in the abdomen to account for death. But on opening the heart, it was evident that the formation of fibrinous coagula had destroyed life. They were firm, decolorised, and adherent, and on the right side they not only obstructed the auricular, ventricular, and arterial openings, but extended far into the subdivisions of the pulmonary arteries, ramifying like the branches of a tree.

CASE XLIV.

TWO ATTACKS OF URETHRAL FEVER.—RECOVERY.

In the month of June 1871 an English gentleman, 43 years of age, of robust and muscular frame, and who had been in India for some years, though not a constant resident of Calcutta, became the subject of treatment for stricture of many years' duration. He was in good health, of temperate habits, married, and regular in his mode of life, much of which had been spent at sea. He informed me that several previous attempts had been made, at long intervals, to dilate the stricture, but without any very satisfactory results, each attempt to pass a bougie or catheter having been followed by fever. He had been able himself to pass No. 4 at times, but apparently this size had never been exceeded. Lately the stricture had been very troublesome and irritable. Attempts to micturate were frequent, disturbing his rest at night, and rendering him very uncomfortable during the day. No. 6 metallic bougie was passed with ease, causing little pain, and only a trace of hæmorrhage. At this time he had been remaining quietly at home for some days, his bowels were regular, and his general health was very fair.

The instrument was passed at about 8 A.M. He was free from

uneasiness all day, took his wonted food, and passed water in a better stream than usual. There was neither hæmorrhage nor pain. At about 5 P.M. he began to feel chilly and uncomfortable. He felt weary; pains in the limbs and body supervened; shortly after a rigor came on, which was rapidly followed by others, each more severe than the last, and when seen a little later they were so violent as to assume the aspect of general convulsions. He spoke with difficulty, and said that although his body, especially his back between the shoulder-blades, was intensely painful, he had no pain in the bladder, urethra, or perineum, and that he had passed urine in a fuller stream than before. Hot fomentations to the back gave him relief, and warm brandy and water with quinine and opium were administered. The convulsions lasting for several hours, were followed by slightly increased heat of the body, with great prostration, and this again by moistness of the skin, which did not amount to free diaphoresis. His pulse soon after the commencement of the rigors became much depressed, rapid, and very feeble, and at times could scarcely be felt at the wrist. His face was congested, and his appearance that of extreme suffering.

He gradually recovered, and regained his strength, but he looked broken and aged by the attack. Throughout, he steadily maintained that he had no pain in the urethra, and that the urine passed more freely and less frequently (though in ample quantity) than before. A herpetic eruption on the lips followed. For several days he continued to take quinine, and was kept fully under its influence; his bowels were regulated, and as good a diet as he could take, with a very moderate amount of stimulants, was given. He had for some years abstained almost entirely from stimulants, but apparently had lived more freely formerly.

In about a fortnight, having recovered, and being encouraged by the improvement already made, he was anxious to continue the treatment, and accordingly at about 9 A.M. an instrument was again passed. On this occasion No. 8 passed as easily as No. 6 on the first occasion. It was observed that there was a certain amount of spasm, and that the urethra grasped the bougie tightly. There was only a slight tinge of blood, and he voided water freely immediately after the instrument was withdrawn. The urine was clear and natural. He said that as the instrument passed through the stricture it gave him rather more pain than on the former occasion.

Ten grains of quinine with opium two grains were given immediately, and were ordered to be repeated in six hours. He passed the day well, and felt no inconvenience (except that the quinine slightly affected his hearing, and the opium made him feel rather drowsy), until about 4 or 5 P.M., when he was again attacked with rigors and vomiting, but not so severely as on the former occasion. There was neither pain nor difficulty about the urethra; a very small quantity of water was passed with ease, and the bowels acted. The rigors were neither so severe nor so prolonged this time; but when they ceased he passed at once into a state almost amounting to collapse, the face being deeply congested, and the pulse intermittent (about 140°), and frequently barely perceptible. His condition was most alarming, and it was feared that he would die. Stimulants, quinine, and diuretics were given; hot fomentations and sinapisms were applied to the loins, for after the first discharge no urine was passed for several hours. He was conscious, but seemed so exhausted that he was unable at times to speak even in the faintest voice.

The following day he remained in much the same condition; the skin was moist, and the temperature but slightly exalted. Towards the evening he voided a small quantity of urine, the irritability of the stomach, which had been very distressing, subsided, and he gradually but very slowly recovered. For many days he remained in a state of great prostration, and when he was sufficiently recovered to enable him to move he was sent out of town for change of air. He had then the gait and aspect of quite an aged man. He continued to take quinine, and had no recurrence of anything like a paroxysm of ague. The urethra on this occasion was apparently slightly benefited; he never had any pain, even on pressure, over the seat of the stricture, and he passed water in an improved but by no means a fully natural stream. The improvement was, however, purchased at so terrible a risk of life, that it was not deemed right to make any further attempt to dilate the stricture.

Remarks.—I have elsewhere expressed my belief that urethral fever is prone to occur in a malarious climate like that of Lower Bengal. I have no recollection of ever having seen it in such a marked and severe form as in Calcutta; for not only does it sometimes supervene after catheterism in tight strictures, where the instrument is passed with difficulty, and the patient's constitution is irritable from the effects of the disease, but also in

slighter cases, and sometimes even when there is no stricture at all, and when the instrument has been passed for other reasons.

The case I have just related seems to support this view. The patient was a healthy man, free from visceral disease, and not, as far as I could judge, previously influenced by malarial cachexia. Though a resident of Calcutta, great part of his time was spent on board ship at sea; he might fairly be regarded to be, as he looked, in good health. He admitted that his stricture was irritable at times, and that every attempt to dilate it, especially in India, had been followed by a certain amount of constitutional disturbance, though far less than on this occasion. He informed me, also, that some of these operations had been attended with considerable difficulty, pain, and hæmorrhage.

There was not the least difficulty in introducing Nos. 6 and 8 into the bladder. There was certainly some obstruction, chiefly arising from spasm, but it was easily overcome, and there was scarcely more than a trace of hæmorrhage. But the treatment was commenced at a most unfavourable season, when malarious influences were exceedingly rife, intermittent fever, neuralgia, and other disorders expressive of their effects being very common. In other respects the season, whilst one of the wettest, had been one of the healthiest ever known in Bengal. The constitutional disturbance that followed each attempt to dilate the stricture was exactly like malarial fever in a pernicious form; and on the second occasion it nearly proved fatal during the state of collapse that rapidly supervened on the rigors. The resemblance of the rigors in the first attack to convulsions is worthy of note, showing how nearly the two conditions are related to each other. Quinine and opium, which were freely administered on the second occasion, directly after the operation, and in anticipation of mischief, seem to have had but little effect. The rigors were certainly less severe than in the first attack, but the subsequent condition was much worse, and for some time his life was in great peril. I have not the least doubt that had the same treatment been followed in England, or in the dry climate of the north-west of India, a certain amount of urethral fever, due to the peculiar influence exerted on the nerve-centres by the operation, would have followed, but it would have been of a less severe character than it was here in the damp and malarial atmosphere of Calcutta. How far the internal and external conditions mutually influence and intensify each other I cannot say; but that they do exert a mutual influence I feel convinced.

Pathologists may see in such cases something to throw light on the subject of the perturbation of the nerve-centres, which in malarious poisoning expresses itself by convulsions in a child, and by an ague fit, continuous or intermitting cold sweats, neuralgia, or other neurotic conditions in the adult.

CASE XLV.

MASKED MALARIAL FEVER.

Report of R. M.'s illness during the voyage from Bombay to Southampton, in the 'Indus.'

On August 2, R. M. had an attack of intermittent fever, to which he had been subject for some years previously in India. After a few doses of quinine the symptoms soon subsided, and for the next three days he complained of great weakness; he was very anæmic. On August 6, at 5.30 A.M., immediately on arrival at Malta, while preparing to go on shore in the early morning before the sun was up, he was suddenly seized with an epileptiform attack, and fell down uttering a cry. This was followed by tonic and clonic convulsions, the latter lasting more or less for over twenty-four hours, during which time he was unable to speak and was perfectly unconscious, crying out loudly at frequent intervals. The temperature during the first twenty-four hours varied from 103° to 104°·4; the pulse, at first imperceptible, soon was felt to be very feeble and rapid, about 120 per minute; pupils dilated, but responding feebly to the light; no paralysis, but very great restlessness. Brandy and beef-tea were given frequently in small quantities. On August 7, at 9 A.M., there was a slight improvement; he was partially conscious and occasionally able to answer a few questions, although he did not recognise any one; he was still very restless and complained of great pain in the head. Temperature 102°, pulse 90; he was able to take food well, which consisted of beef-tea, milk, and a little brandy occasionally. From this time he gradually improved, and on the morning of the 8th instant was quite sensible, with a normal temperature. He did not remember anything that had taken place during the previous forty-eight hours, and complained of slight pain in the head occasionally, and great stiffness in the limbs; he is now improving daily, but is very weak and anæmic.

The treatment at first consisted in giving twenty-grain doses of bromide of potassium at frequent intervals, which had a very beneficial effect in checking the restlessness and convulsions;

cold applications were applied to the head, and heat to the feet; later, tonics and quinine were given.

The above is an abstract of this case furnished by the medical man who attended the patient on his way home. On examination in London he was found to be anæmic and debilitated; there was a cardiac hæmic murmur, and the pulse was quick and feeble urine normal. This is probably a case of masked malarial poisoning.

He had been in India since 1863, but came home from 1875 to 1876. He had served some years in the malarial climate of Assam. He is forty-two years of age, and his previous medical history is good.

LECTURE III.

IN my last lecture I described paroxysmal fevers of malarial origin, and also certain morbid conditions depending on the same causes. I now proceed to consider fevers which are neither regarded as primarily malarial nor are of a paroxysmal character, and in their simple forms are due to ordinary causes such as produce febrile disturbance anywhere; also others which are so closely assimilated to the malarial remittents and specific fevers, that it is difficult to differentiate them, and obviously impossible to consider them under such designations as *febri-cula* or *ephemeral fever*.

As to the precise nature of their cause we know no more than we do of that of malaria itself, nor are we assured that there is an essential difference, unless it be that a predominance of animal organic decomposition and effluvia gives a distinctive character. But the importance and frequent severity of these fevers and their etiological affinity to the remittent forms, render it expedient to place them under a distinctive heading. I would therefore suggest that the following classification might be adopted, which, while fully acknowledging the other forms, recognises the existence of a type of fever of a continuous or sub-continuous nature, which, by some authorities, is ascribed to the combined operation of a double agency; by others it is attributed to malaria alone, or to changes that result in a transformation of type, especially when certain visceral, *i.e.* hepatic and gastro-intestinal, complications occur.

I would propose the addition of endemic enteric or continued fever, but could not insist on the term

‘endemic’ if another could more appropriately indicate the type of fever to which I refer. The arrangement might be as follows :—

Continued Fevers in India and the Tropics.

- (a) Ephemeral or febricula.
- (b) Ardent or thermic fever.
- (c) Endemic enteric or continued fever.
- (d) Specific typhoid fever.
- (e) Dengue.
- (f) Typhus fever.
- (g) Relapsing fever.
- (h) The adynamic contagious fever known as Pali-disease, mahamurri, or Indian plague.
- (i) The specific yellow fever, should it ever appear in India, as distinct from the severe form of bilious remittent which it closely resembles.

This addition to the present nosological arrangement would give greater precision to registration and lead to more definite conclusions as to the true etiology and pathology of diseases about which it cannot be denied that there is at present some confusion in reference to their true nature and causation—being regarded as remittent, continued, or specific enteric, according to the views of different observers.

Without for a moment presuming to question the diagnosis, I venture to think that there may be a too rigid adhesion in India to views of disease as it occurs in this country, and that sufficient allowance is not always made for the influence of new conditions whereby it may be modified and made to assume features strange to it, in temperate climates.

Some medical officers of experience see reason to modify their views of disease after experience in India, and it is fortunate for the future of tropical pathology and medicine that they do so; as a too exclusive application of theories which are strictly appropriate here, will not always be so in India and in the tropics.

The late Dr. Babington, in an address to the Epidemiological Society, made the following remarks, which I cannot help thinking are applicable to this point :—

‘In the infancy of geology, many phenomena observed in the arrangement of the earth’s crust, as it is found on this island, were supposed to furnish fixed laws, and thus gave rise among our philosophers to divers ingenious generalisations. But when these same philosophers had, from the establishment of universal peace, the opportunity of taking a wider range and of studying the earth’s structure, not in this country alone, but over the whole surface of the globe, they discovered in many instances that what they had supposed to be general laws were after all only exceptional cases. We require, therefore, in a study of disease, as of geology, a wide field, in order that we may found theories on a sufficiently broad basis to avoid the risk of coming to partial and erroneous conclusions.’¹

Or, as Dr. Morehead says in a letter to me—

‘Disease in India is not disease in England, and a catholic science of medicine can only be created by the harmonious action of labourers in varied climates and conditions of people, and nothing can be more fatal to this issue than assimilation by official control.

‘The manner in which Bryden has shown the relations of enteric fever in India to the age and service of soldiers has, I think, had the tendency to suggest, what he cannot have intended, that all the fevers of the young soldier are enteric. This, as you remark, would be absurd. Take, for example, the record of the contrary in Sections 2 and 3 of Chapter ix., p. 162, of the second edition of my “Clinical Researches.” Febricula and ardent continued fevers ought to be, with the sanitary system of the present, much less common than they were in the past; but you cannot destroy the fact that they have been, and will be again, when the causes are allowed to become operative.’

¹ Address to the Epidemiological Society, by Dr. B. G. Babington, December 1850.

Ephemeral Fever or Febricula.

A mild form of simple fever is of frequent occurrence in India, which is due to ordinary causes, such as changes of temperature, excesses in eating or drinking, fatigue, excitement, disordered secretion, or functional derangement of the abdominal viscera. It is most frequent in the hot seasons, but may occur at any time. When it happens in persons newly arrived in the country it is probably free from any malarial taint, though the onset may resemble ordinary ague. In natives and old residents these simple attacks of ephemeral fever or febricula *are* probably of a malarial nature, and if not checked may assume the character of an ordinary intermittent. The symptoms are headache, foul tongue (white, with red edges), disordered bowels, nausea or sickness, high temperature (104° – 106°), preceded by chills, rigors, malaise, muscular pains, and followed by diaphoresis. In plethoric or intemperate persons the reaction may be severe, with high fever and delirium; to which may be added congestion of the liver or gastro-intestinal mucous membrane; but generally the characters are simple, and in otherwise healthy subjects readily yield to treatment which consists in free relief of the bowels, in some cases an emetic, diaphoretic medicine, tepid sponging, a restricted diet, and rest; with this the patient is generally restored to health in a few days. Most people soon after arrival in India have fever of this character, and as a general rule with the most ordinary case it is soon recovered from. The change of life, the heat, the functional derangement of stomach and liver, the irritation of musquito bites, want of rest, &c., all tend to develop a condition of disturbed innervation and nutrition, and blood-contamination by retention of effete products and imperfect elimination and assimilation, that find expression in a febrile condition. Dr. Macleod, of Calcutta, expresses it tersely as follows:—‘There is a hot-weather fever which I call the punkah fever, due to checked skin depuration; another

in the rains, due to deranged liver action ; another in the cold weather, caused by too much work being thrown suddenly on the kidneys. These fevers do not require quinine, and yield to action on the bowels, skin, kidneys, or liver. They are apt to assume a remittent type, and *enteric complications are not uncommon*. In fevers of this form emetics are often of service, and salicylates, by stimulating skin, liver, and kidneys, are useful.' In this fever there is nothing peculiar unless complications arise, when it may be prolonged and assume a more severe condition.

It is needless to say more about treatment than that aperients, saline diaphoretics, the cold or tepid bath, restricted diet, and rest, with a few doses of quinine (this is always desirable), are generally sufficient to restore the patient to health. Few escape, and to Europeans it is an acclimatising process.

Ardent or Thermic Fever.

This is a much more serious disease and varies in intensity from simple to intense fever, or reaching its maximum, to sunstroke—the exciting cause being solar or artificial heat. This form of fever occurs chiefly when the heat is most intense in the months preceding the rains. During the hot winds it is frequently very fatal even to natives, who succumb to a rapid and acute form of it, known as heat apoplexy, hot wind stroke, or sunstroke. This may be immediate, or a high state of pyrexia leads up to it less rapidly.

The effects of over-heating the living body have been described by Claude Bernard, Lauder Brunton, and others. Professor H. C. Wood, in his recent magnificent monograph on fever,¹ gives the results of experiments which show that when above a certain degree, heat, by inducing vaso-motor paralysis, causes intense pyrexia, which if not

¹ *Fever ; a Study in Morbid and Normal Physiology*. Smithsonian Contributions. Vol. XXIII. Washington. 1880.

mitigated, rapidly destroys life by causing failure of the respiratory centre. These researches confirm the fact that high temperature is capable of causing most fatal nutritive disturbances, and that thermic fever does the same. The peculiar odour and offensive perspiration, the altered or suppressed urine, the frequent watery, offensive, involuntary evacuations, the broken-down crases of the blood found after death, are all indications of the profound influence excited by excessive temperature.¹

Heat may cause simple continued fever, but if aggravated the symptoms become more urgent, and the temperature may rise to 106°–108°, or higher, when life is in the greatest peril. The predisposing causes are previous illness, debility, intemperance in food or alcohol, constipation, bilious derangement, imperfect breathing from over-crowded rooms and barracks, ill-ventilation, or defective perspiration. The vitiated and over-heated blood acting on the nerve centres paralyses the heat-controlling centre; there is great rise in temperature, producing congestion and death if the person affected is not relieved.

The effects of heat in causing fever, or those more serious conditions known as sunstroke, are as follows:—

There is syncope or exhaustion caused by the action of the direct rays of a powerful sun; the centres are affected, respiration and circulation fail, and death may result.

Over-heating of the blood and nerve centres, either by direct exposure to the sun's rays or to high temperature, may cause vaso-motor paralysis and hyperpyrexia; respiration and circulation fail, and asphyxia follows, or fever, which may become intense and lead to sunstroke. Recovery is often incomplete, owing to structural changes, which give rise to a variety of symptoms of a grave character.

¹ 'External heat applied to the body of a normal animal (or man), so as to elevate the temperature, produces derangement of the functions of innervation, of circulation, of nutrition and secretion, similar to those seen in natural fever, the intensity of the disturbances being directly proportionate to the rise of temperature'—Wood, on *Fever*.

Simple exhaustion and syncope may occur during great fatigue or over-exertion, or when there is depression of vital power from any cause during exposure to a high temperature. This may be observed in the case of the engine-room men of the steamers in the tropics, when the temperature in the vicinity of the furnaces where they are employed rises to 120° and upwards; or in that of men who are exposed to the intense heat and light of the sun's rays, which taking effect on the head, neck, and body, produce a condition like shock. In the first case the skin is pale, cold, and moist, the pulse feeble. Death may occur from failure of the heart, but recovery is frequent; or asphyxia and apnoea may supervene after premonitory symptoms of depression and weakness, during exposure of the head and spine to the direct rays of a powerful sun, when the atmosphere is much heated, and the nervous energy is depressed by fatigue, illness, or dissipation. The respiratory centres are overwhelmed by the sudden rise of temperature, and respiration and circulation fail. When death takes place suddenly it has been ascribed to rapid coagulation of cardiac myosin. This, however, is probably a post-mortem change; the heart's action having been brought to a close by heat, it having been shown by Claude Bernard and Lauder Brunton that the effect of a very high temperature on animals is first to accelerate, and finally to stop the heart, in a state of tetanic contraction.

Recovery is sometimes tedious and imperfect, ending in serious impairment of health or intellect.

In other cases there is ardent fever, the body generally being intensely heated. This may occur, independently of the action of the direct sun's rays, at night—a condition which may come on in ordinary health, but is more likely to do so in the debilitated, or it may complicate any other disease, and especially malarial fever. It may also occur in the shade, in a house or tent, especially in persons who are depressed by fatigue, bad air, over-feeding, alcoholic stimulants and consequent depression,

want of rest or illness, and notably when the air is impure from overcrowding, or from insufficiency of cubic space.

The body temperature may rise gradually or rapidly to 108° or even higher; there is dyspnoea, hurried respiration, restlessness, and a pungently hot skin, which is sometimes dry, occasionally moist. The pulse varies; in some it is full and labouring, in others quick and jerking; the head, face, and neck are livid and congested; the carotid pulsation very perceptible; pupils, at first contracted, dilate widely before death. Coma, stertor, delirium, convulsions, frequently epileptiform in character, with relaxation of sphincters, and suppression of urine, are the precursors of death by asphyxia, and there may be cerebral hæmorrhage.

A large proportion of the fatal cases among Europeans in India is so caused. Partial recovery may be followed by relapse and death; or secondary consequences may destroy life or impair health and intellect at a later period. The premonitory symptoms of this form of the disease appear some hours or even days before the dangerous conditions supervene. There may be general malaise, disordered secretions, profuse and frequent micturition, restlessness, insomnia, apprehension of impending evil, hurried and shallow breathing, precordial anxiety, gasping, giddiness, headache, occasionally nausea or vomiting, thirst, anorexia, and fever, which soon amounts to fervent heat of skin; the surface may be dry or moist, the pulse varies. These conditions gradually become aggravated, frequently being worst at night, and the patient may pass into a state of unconsciousness and die.

Ardent fever of this character may supervene on ordinary ephemeral fever, heat alone, especially when the atmosphere is loaded with moisture so as to prevent evaporation from the person, being the real cause. Malarial and hygrometric conditions have no special influence beyond that which they exert in predisposing the person to suffer.

The dry atmosphere of Upper India, with its hot

winds, is better tolerated than the damp atmosphere of Lower Bengal or parts of Southern India, though the temperature is lower. Hot *dry* air favours evaporation, and thus keeps the body cool, whilst in damp air, as evaporation decreases, the natural cooling power is greatly diminished.

Vigorous, healthy persons of moderately spare frame, with sound viscera and temperate habits, can sustain a great amount of heat if the atmosphere be pure and moderately dry. Acclimatisation has some influence in conferring toleration. Fresh arrivals in India are more prone to suffer than those who have become accustomed to the climate and have learned how to protect themselves. It is well known that a native can bear an amount of sun on his bare head and naked body with indifference, almost pleasure, that would prostrate a European. But when the temperature rises above a certain standard all succumb, and natives of India suffer and die like others.

The extent and duration of toleration of heat ¹ depend

¹ In the Fourteenth Annual Report of the Sanitary Commissioner with the Government of India, 1877, it is stated that 235 cases of heat apoplexy and sunstroke occurred in the army in India, of which 70 were fatal. The admission rate, 4·1, is almost the same as in 1876, and exactly the same as in 1865. The death rate, 1·22, is below the average of the last six years. Of the 235 cases, 189 occurred during the four months of May to August. The disease was widely spread. It gives rise to a fatality of 12·2 per cent. in the first and second years of the European soldier's service in India.

The deaths per 1,000 strength :—

SERVICE.

1 to 4 years	5 to 7 years	Above 7 years
1·48	1·05	1·50

AGE.

Under 25	25 to 29	30 and upwards
·65	·99	2·33

much on the vigour of constitution and actual state of health. The refrigerating powers of the body, in health, enable it to support a temperature considerably above that of the blood. In the hot winds little inconvenience is felt so long as perspiration is free, but when that fails, suffering ensues, and the danger is great.

But of those who recover, or rather who do not die, many are permanently injured, and remain invalids for the rest of life, which is frequently shortened by the changes induced. These may be due to obscure cerebral or meningeal alterations, which affect the sufferer in various degrees of intensity, producing irritability, impaired memory, epilepsy or epileptiform attacks, headache, mania, partial or complete paraplegia, partial or complete blindness, extreme intolerance of heat, especially of the sun's rays—rendering a person quite incapable of serving in hot climates or of enduring any exposure to the sun. Or there may be a gradual termination in complete fatuity, dementia, or epilepsy, perchance both; chronic meningitis, with thickening of the calvarium, which may account for the intense cephalalgia; or, in a lesser degree, disordered innervation and general functional derangement, which seriously compromise health.

In cases where death has occurred suddenly, as from syncope or shock, there is no very remarkable morbid change. The heart may be firmly contracted, but not always so, for it is often flaccid. The lungs, brain, and membranes may be congested, but they are sometimes quite the reverse. The venous trunks, especially those of the abdomen, and the right side of the heart itself, may be filled with blood, which is dark, grumous, often partially coagulated, and effused in patches of ecchymosis, rendering the body rapidly livid. The coagulability of the blood is impaired, and it is deficient in oxygen.

In death from ordinary cases of thermic fever the lungs are often (not always) deeply congested; the heart is firmly contracted by coagulation of myosin, and the whole venous system is engorged. The body, even before

death, may be marked by petechial patches and extensive livid ecchymosis. The blood is generally more fluid than natural, and may be acid in reaction. The globules are sometimes crenated and have a diminished tendency to form into rouleaux. The body for some time after death retains a high temperature. When first opened, the viscera and interior feel pungently hot. Rigor mortis comes on very rapidly. The brain and membranes may be congested; in some cases there are evidences of cerebral hæmorrhage and serous effusion in the ventricles.

In cases of simple exhaustion, remove the person to a cooler place, if possible. Give a douche, but not too prolonged, or it may depress; rouse, and gently stimulate; remove tight and oppressive clothing; apply ammonia to the nostrils, &c. Rest and avoidance of exposure to over-fatigue or to great heat are necessary.

When the person is struck down suddenly by sun, remove him into the shade, and let a douche of cold water fall from a height on his head and body, the object being to reduce temperature and to rouse by reflex action. During the assault on the 'White House picket,' at the capture of Rangoon in 1853, numbers of men struck down by the fierce April sun were so treated; only two, who had been bled, died. Sinapisms may be applied to various parts of the body, legs, abdomen, &c., and stimulating enemata may be useful.

When I say such cases recovered, I refer to the reaction produced at the time. In some there were consecutive symptoms of fever, headache, &c.; and could we trace their subsequent history, it is probable we should find that complete recovery never occurred. If recovery be incomplete, and followed by indications of intra-cranial mischief, other treatment of a more active character may be needed. Future exposure to the sun should be carefully guarded against, and, unless recovery be complete, the sufferer should be removed to a cooler climate, and protected from excitement of mind or body, whilst the greatest care is taken to avoid all errors or excesses of diet and stimulants.

In thermic fever, the object is to reduce temperature as speedily as possible before tissue changes have been caused. As the hyperpyrexia is due not only to the direct action of heat on the nervous centres, blood, and tissues, but to the vaso-motor disturbance, remedies that may influence this are indicated. The use of quinine by hypodermic injection has been thought to produce good results by reducing temperature.

Bleeding has been abandoned except in rare and peculiar cases. There are cases in which it may be necessary in order to avert suffocation, but they are, I think, exceptional. Where it has appeared at first to give relief and to mitigate the symptoms, the improvement has generally been transient, and followed by relapse into a more dangerous and fatal condition.

The treatment generally consists in the application of cold by affusion or by ice, taking care not to reduce the temperature too low. A thermometer in the axilla, mouth, or rectum, will keep one informed in this respect, and danger would attend continued depression of the temperature below the normal blood heat. The bowels should be relieved, and cooling medicine given. The earliest and most severe symptoms having subsided, the febrile condition that follows is to be treated on ordinary principles; and the diet must be carefully regulated. If after severe cases, as improvement progresses, symptoms of intra-cranial mischief supervene, iodide of potassium and counter-irritation may be of service; and removal to a cooler climate is essential. As a general rule it is desirable that the sufferer should not return to a hot climate, and he should be guarded against all exposure to heat, overwork, and anxiety of any kind. In simple cases of sun fever, where the reaction is not excessive, the treatment is that of ordinary ephemeral fever.

Continued Fever.

Writers on Indian and tropical disease have described a form of continued fever liable, like remittent, to be

modified by visceral complications, and to have a fatal termination, post-mortem examination revealing pathological changes of various degrees of importance. It is attributed to climatic causes, and the circumstances attending life in tropical or sub-tropical regions, such as heat, atmospheric vicissitudes, terrestrial emanations, personal habits; and no very distinct characters differentiate it from remittent when it has assumed a continued form. Twining, Annesley, Martin, and others refer to such a fever, and generally, I think, they regarded it as a variety of malarial fever, in which perhaps there is little difference of opinion. But it is necessary to distinguish it from specific continued fevers, with which it may be confounded. In typical cases of remittent, the diagnosis is clear enough, but in many others it is difficult, if not impossible, for the characters of the temperature curve vary so little that it is not possible to deduce from them any certain differential points of diagnosis. It would appear that other fevers peculiar to India may assume this condition, especially if not dealt with properly at first, and that when visceral complications occur, they are generally, if not always, the precursors or concomitants of the change of type.

Enteric Fever.

It was not until the year 1853 that attention was called to the pathological changes in the intestines of persons dying of fever in India, when it began to be suspected that certain protracted and fatal cases were due to a disease identical with the typhoid fever of England. Careful observation of the morbid appearances after death, and the symptoms and progress of the disease during life, led observers in India to believe that the diseases were one and the same, modified, it might be, by climate and the influence of malaria. Further observations in different parts of India established this view, and in a few years typhoid fever became fully recognised as a prevalent and fatal form of disease, especially among young and

susceptible Europeans, a class notably represented by the soldiers of our army in India.

Annesley has left it on record that he had never remarked any appearance of fever from a specific or contagious source in India, and that, although believing in the influence of infection as regards the continued adynamic fever of temperate climates, he had never, during an experience of thirty-seven years in India, observed fever to proceed from contagion in that part of the world. The fevers, therefore, of India, and he believed of warm climates generally, are the effects of exhalations from the soil and of vicissitudes of season, the former especially occurring in predisposed constitutions. The types and forms which these fevers assume are entirely dependent upon the activity of these causes, in relation to the conditions of their subjects, and various collateral circumstances occurring about the time of their invasion; and fevers in India vary in every possible grade and form, from the slightest febricule or ephemeral attack to the most malignant type, which is tantamount to saying that all fever in India is climatic, and that specific contagia are altogether excluded—the course and characters of fever depending on local determination and complication, and external influences, the originating cause being one and the same, though varying in activity and intensity. I think it is a general impression among those who have studied and treated disease in hot climates, that Annesley rightly enough expressed the importance of this etiological question; and few will dissent from his views as far as they apply to much of the disease in question, that some of the continued as well as the paroxysmal fevers are due to what may be described as climatic causes.

But great advances have been made in our knowledge of the nature of fevers since he wrote. It has been clearly pointed out that in India fevers arising from specific contagion do occur, and it is now well known that relapsing, typhus, and typhoid fevers are Indian diseases.

It is within the period of my own service in India that

attention was first directed to the existence of typhoid fever in India. Before that time it had not been noticed, but it is now fully established, and appears in the Sanitary Report as the chief fever death-cause among our young soldiers in that country. It would, however, be as reasonable to say that it had *not existed in England* before you, sir, and others defined it to be a specifically distinct disease, as that it did not exist in India before Assistant-Surgeon Scriven, of the Bengal Medical Service, pointed out its existence in that country, when, guided by the light thrown on it by British research, he separated it from remittent in India, as in England it had been separated from typhus. The honour of this important step in fever pathology is, as far as I know, clearly due to Scriven, and his views were confirmed after he had promulgated them in 1853, by Dr. J. Ewart, a Fellow of our College, and by the late Dr. E. Goodevear, who published a valuable clinical lecture on the subject in the 'Indian Annals of Medical Science' of January 1859, in which he pointed out its identity with typhoid fever in Europe. Since then it has been observed all over India, and is now incorporated in the statistics for the whole of India. But these gentlemen did not discover a new disease; their merit consisted in pointing out one already existing, but which had hitherto been overlooked as distinct from other diseases with which it had been associated and confounded.

Dr. Joseph Ewart was, as far as I know, the first to have observed the existence of specific typhoid fever in the natives of India. This discovery was made quite independently of Scriven's first discovery of the disease among Europeans in India.

Annesley, Twining, Morehead, and others had long ere this pointed out the frequency of typhoid symptoms, diarrhoea, enteric ulceration, and other phenomena characteristic of adynamic types of fever.

Annesley says¹:—'The fevers of warm climates, especially as observed in the Eastern hemisphere, seldom

¹ *Diseases of India*, p. 535.

go through their entire course without evincing a predominance of morbid action in some viscus or texture, most frequently those seated in the abdominal cavity and in the cranium. I do not, however, consider that the increased disease in certain localities ought to be viewed as the immediate cause of the febrile excitement, or, in other words, that fever is merely general disorder supervening on disease of a particular organ; but, on the contrary, that the exciting causes of fever produce disorder of the frame generally, which, owing to the predisposed state of certain viscera or textures, occasions a prominent derangement of them; and that if this superinduced disorder be allowed to proceed, it often aggravates the general fever, and rapidly terminates in organic lesion.

‘Amongst the most early local affections which appear in the course of intertropical fevers is an inflammatory state of the mucous surface of the stomach and duodenum.

‘In the progress of those fevers in which these are prominent symptoms, especially in the bilious remittent and bilious inflammatory continued fevers, and in many of those which assume characters of a malignant kind, the inflammatory state of this part of the digestive mucous surface exists in a more or less aggravated form, and not unfrequently extends to the internal surface of the small intestines, and even, in some cases, to the large bowels. This extension of the inflammatory action to the small intestines is indicated by tumefaction and tenderness of the abdomen to pressure made about the umbilicus, by a sense of inward soreness, or heat in this situation, and by an irregular state of the functions of the bowels, attended with occasional sickness, and a frequent, scanty state of the alvine discharges, approaching to diarrhœa, and sometimes to an intermediate state between diarrhœa and dysentery.’

He also says:—‘Marks of disease of the small and large intestines are generally confined to their internal tunics. The duodenum, jejunum, and ileum, especially the duc-

denum and termination of the ileum, very frequently are diseased in their mucous surface, which is inflamed in patches, sometimes covered with a muco-purulent secretion, and studded with small ulcerations, particularly the termination of the ileum. Occasionally the mucous surface is of a brick-red or purplish shade of colour, apparently ecchymosed, and covered with a bloody sanies, and readily detached from the subjacent texture. In several cases, the ulcerations, which sometimes are large and far apart, at other times small and agglomerated, especially the former, have nearly penetrated the tunics of the intestines, and in a very few cases I have observed this occurrence actually to have supervened, the contents of the bowels being partly effused into the peritoneal cavity, and having produced peritonitis.

‘Marks of inflammatory action are occasionally met with in the peritoneum, omentum, and mesentery, in all the forms of fever; and in protracted cases of the remittent and intermittent types, especially those in which the liver and spleen have been obstructed or otherwise diseased, considerable effusions of a serous fluid into the cavity of the abdomen are not uncommon.

‘In these cases the peritoneum presents either a sodden appearance or congestion of the veins. In many of those cases, also, the mesenteric glands are enlarged, of a light colour, and hard consistence. Diseased appearances of the mesenteric glands are not associated alone with the dropsical effusions, as they are frequently observed when no such effusion is present, and when the mucous surface of the bowels is diseased, and the liver and spleen enlarged, and otherwise changed in structure.’

Twining in 1842, describing what he called the congestive fever of the cold season, says:—‘There is often much congestion at the root of the mesentery, and in the fat and cellular structures surrounding the duodenum, where it is bound down across the spine. In a few rare instances where patients have died after a protracted fever of this sort, superficial ulcerations of the mucous

membrane of the small intestines were found. I will not venture to assert that the ulcerations above alluded to ought to be considered as causes of the fever of the cold season; and my reason for not deeming that pathological condition a primary affection existing at an early period of the disease is, that active purgatives may be repeated daily for a long time at the commencement of this fever without producing irritation—in fact, they almost always afford relief; whereas we do sometimes find that active purgatives produce a degree of intestinal irritation at a late period, and when a fatal termination takes place afterwards, ulcerations of the small intestine are found in these subjects. If some extended observations should prove that these ulcerations of the small intestine exist *generally in the cases* which terminate fatally, and that such a pathological condition is rarely met with in the inspection of subjects that have died of other descriptions of fevers in Bengal, I should be inclined to adopt the opinion that a *peculiarity* of the disease would be thus ascertained, which, combined with the exclusive prevalence of this fever in the cold season, its insidious invasion, obscure symptoms, slow progress and protracted course, attended with prolonged stupor and delirium, and the organic changes at its later stages, might establish a resemblance to some modifications of European typhus; although the resemblance be not strictly correct in all its details.' The general characters of the fever he describes present a similarity to European typhoid; it is evident that it is no new discovery, and that the bowel or other lesions did not escape notice.

Sir R. Martin, speaking of the congestive continued fever of Bengal, says:—'In neglected cases we find hepatic abscess and sometimes ulceration of the mucous digestive surface. *The latter* I found to be very prevalent among the labouring classes of natives whom I had to treat at the Native Hospital of Calcutta, on account of neglected fevers of from *fifteen to twenty days'* duration, and a large proportion recovered.'

It seems obvious from these references that fever with intestinal ulceration and other symptoms characteristic of enteric fever were observed in India before 1853.

Dr. Morehead, in the second edition of his 'Researches on Disease in India,' p. 160, remarks that the observation of a case, together with the report of Scriven, Ewart, and Goodeve, removed the doubts he had previously entertained as to the existence of typhoid in India, and says:—'The investigation will require to be prosecuted with much care in order that the tendency so common in medical research to exaggerate the importance of new subjects of inquiry to the neglect of established truths may be sufficiently controlled; and that it is to be recollected that disease of Peyer's glands, either in the stage of turgescence or ulceration, is not peculiar to typhoid fever only, for it occurs in cholera, in protracted diarrhoea, and in acute muco-enteritis, or as an *occasional complication of remittent fever*, and is a frequent one of *phthisis pulmonalis*.'—From which it follows that we are not justified in deducing the existence of specific typhoid fever from the mere character of the post-mortem appearances, which require to be interpreted by the symptoms which have been present during life in order that they may be correctly understood.'

In a letter to me he observes:—

'I, with you, have never been satisfied with Budd's exclusive theory or with the evidence which he advanced. Murchison's general sewage views always seemed to me to come nearer the truth; but I am not quite certain whether he adhered to them in his latest writings. I am entirely with you in thinking that Peyerian ulceration is by no means necessarily the product of one cause, and I would add, or in association with one set or order of symptoms. On this point I gave a warning in the second edition of my book; and it is from neglect of this pathological fact that much of the confusion in India has arisen. . . . You incline, if I mistake not, to the

opinion that there are in India cases with the symptoms and lesions of European enteric fever, which cannot be traced to a faecal cause, either on Budd's theory or Murchison's, but which must be traced to climatic or other ordinary causes. I do not dispute it; nay more, I shall not be surprised if it prove so. All I say is, that I have seen no evidence that satisfies my judgment; nay more, I do not think that there has been clinical investigation of a quality and to an extent to settle the question; and that therefore it should be relegated to India for further and better clinical inquiry.

'I gather that your idea is that the symptoms and lesions of enteric may be caused, and are in India, by climatic causes. I will not dispute this, and I am very much disposed to share in your anticipations on this point. But the question at this moment seems to me to stand thus: Insist upon precise clinical reports of enteric irritations, with special attention to the question of diagnosis and to causation. This ought to elicit whether they are all or even in great part explainable on the sewage etiology; if they are not, then this dogmatic etiology will be disproved by the observation of the disease in its geographical relations. This I believe will be the result; but this is for the future, and I almost think that it will be better to let this course be quietly followed than to *ante*-discuss the question before the facts are ripe to sustain it.'

Dr. John Macpherson states that somewhere about 1851 Assistant-Surgeon Lee, just from home, made several post-mortems with him at the General Hospital of cases of fever of the hot weather, occurring chiefly among European seamen. Mr. Lee was surprised to observe ulceration of Peyer's patches, and was immediately reminded of cases of dothinerteritis which he had just been seeing in Edinburgh. Dr. Macpherson had not been in the habit of examining the small intestines minutely in fever cases, it being the received doctrine that they were *not much*

affected in tropical fevers. The fevers in which the ulceration was discovered did not appear to him to differ from fevers which he had been treating for many years, except in so far as he was accustomed to see the types of fever vary considerably in different seasons, the marked variations being—

In the amount of head symptoms.

Intensity of lumbar pain.

Gastric irritability.

Presence or absence of diarrhoea, the case not being amenable to quinine, being protracted, and what used to be called a typhoid state supervening, with occasional redness of the fauces, and occasionally a roseolar rash, of which much was not made.

As a general rule, fevers were more acute in the hot weather, and more protracted in the cold season.

There was in these cases much more pyrexia, and the course was more acute than he understood to be the case in European typhoid. He therefore did not regard the cases as typhoid, and sought for no new cause to account for fevers which appeared to him to present little novelty in their general symptoms. He certainly never thought of fæcal poisoning, nor of any specific cause for what he considered to be varieties of the ordinary fevers which he attributed to exposure, climate, and season.

My own experience, so far as it goes, coincides with that of Dr. Macpherson. In or about 1854 my attention was arrested by a case of fever at Lucknow in the person of a young French gentleman of between twenty-eight and thirty, who died after a protracted fever of more than three weeks' duration, attended with diarrhoea, hæmorrhage from the bowels, iliac gurgling, tympanites, stupor, sordes on the tongue and teeth, and collapse evidently supervening on perforation. This gentleman had been exposed to malarial influences, and the fever was regarded as climatic, for there was no reason to suppose that he had been exposed to the influence of fæcal poisoning, though of course it is impossible to prove a negative. I then

thought of the possibility of malarial fever assuming the enteric form. Dr. Maclean, C.B., the distinguished Professor of Medicine at Netley, says :—‘ So far back as 1838 I treated fevers in Secunderabad, in the Deccan, and in China as far north as Nankin, extending over more than twenty days, with bowel complications. The mortality exceeded that of fevers distinctly malarial, and they were not amenable to quinine freely given ; death from hæmorrhage from the bowels was frequent, and the intestinal lesions were those we now recognise as characteristic of enteric fever.’¹ I think it is needless to cite more evidence of the existence of fever with typhoid symptoms and Peyerian ulceration antecedent to the year 1853.

Dr. Gordon, C.B., late Chief of the Medical Service in the Madras Presidency, says :—‘ In fevers as I saw them in British soldiers, enteric complications, including ulcerations precisely like what occur in specific fever in this country, occur in fevers (in India) that cannot be traced to anything pythogenic or otherwise specific. If a non-specific fever in the tropics occur in a young delicate lad, it will almost to a certainty become complicated sooner or later in its course by diarrhoea or dysentery, and ulceration will be found in the small or large intestines, Peyer’s glands included. Is it meant to call it “ enteric,” in a sense that it is pythogenic ? If so, I believe that the designation is wrong.’ I do not gather from Dr. Gordon’s opinions, as expressed in many reports and papers he has written, that he denies the existence of specific typhoid fever in India, or that he considers it as a new disease ; but rather, that he insists on the necessity of sifting all cases, and of examining closely into their history, with the view of ascertaining if cases recorded as enteric, thereby meaning specific fæcal enteric fever, may not have been of malarial or climatic origin.

Dr. Chevers says :²—The question, Is enteric fever at

¹ Dr. Maclean’s observations prove that fever with enteric ulceration existed previously to 1851.

² *Medical Times and Gazette*, September 20, 1879.

present a common or a rare disease in India? is certainly one of the most perplexing, as it assuredly is one of the most practically momentous questions with which medical men in that country have to cope. It being a plain fact that if we, encountering a case of *paludal remittent, with bowel complication*, insist upon calling it true enteric fever, and treat it as such, withholding that free and steady use of quinine which is the only remedy, that case will almost inevitably end in death. Dr. Bryden assuredly did a good and very needful work in giving enteric fever a place in the register of cases; but after admitting this, I confess my unwillingness to admit that we have proof sufficient to convince us that 571 European soldiers of the Indian army died in six years of true enteric fever.' In a letter to me Dr. Chevers says:—'There may be follicular ulceration, first tubercular, second dysenteric; but whenever I have found such ulceration in a case of fever, the other features have been those of true enteric fever.' Dr. Chevers further says¹: 'If there be two forms of Indian enteric fever, there may possibly be two sets of intestinal lesions. My own English and Indian experience, however, shows no change in these lesions,' and he gives the following excellent advice to young medical officers: 'Approach each fever case, where the disease assumes a continued form, in a spirit of vigilant inquiry, and never rest satisfied until you have sufficient grounds for determining whether it is one of remittent or true enteric.'

Deputy Surgeon-General Dr. Alexander Smith, speaking of fever in India, says, in 1873:—'With the setting in of the rains in June or July the fevers assume more of the low remittent and continued types, running a much slower course, and showing in an unusually marked degree, especially in the latter form, in a majority of instances, the bowel complication held by medical writers to be characteristic of so-called "enteric" or "typhoid" fever.'² He denies the specific origin of enteric fever, and attri-

¹ *Medical Times and Gazette*, September 27, 1879.

² *Fever and Cholera*, p. 15, 1876.

butes it to general and climatic causes; and asserts that for the germ hypothesis of typhoid fever there is no trustworthy evidence.

Dr. Wall, of the General Hospital of Calcutta, writes to me:—‘I believe that a large proportion of cases returned as typhoid fever have no right to that name. If a man die in India after having an elevated temperature, and an ulcer can be found in his intestine, the case is at once called typhoid. But it takes a great deal more than an intestinal ulcer to make a typhoid fever. I have seen many cases that could not with certainty be referred to any type of fever, but which had on the whole more resemblance to remittent than any other, and which were found after death to be coincident with intestinal ulceration, but an ulceration distinctly not typhoid. It was an irregular ulceration, by no means selecting the site of Peyer’s patches, and very often encircling the intestine; and my experience is that this form of ulceration often occurs in cases that would better bear the name “remittent” than anything else.’

Dr. MacConnell, the very able Professor of Pathology in the Medical School of Calcutta, writes to me:—‘As regards typhoid or enteric fever in this country, and its etiology, I am inclined to believe that the evidence of a specific poison is not nearly so generally available here as in Europe, and that probably climatic influences, plus want of proper sanitation, gives rise to not a few cases in India.’ He continues:—‘There is the great difficulty in diagnosis. In all the cases that I have seen here and verified by post-mortem examination, neither the course of the fever nor the range of temperature has been at all typical, and the presence of rose spots or of any specific eruption has been quite exceptional. Of course it is more difficult to see rose spots on the dark skin of a native, but they have been looked for carefully and repeatedly, and yet not found.

‘Malarial agency seems to modify the whole course of the disease, and thus one great help in diagnosis at home,

viz., the *diurnal range of temperature*, is wanting to us out here. Murchison says "that the eruption of the lenticular spots is perhaps the only reliable distinctive mark of pythogenic fever," *i.e.* between it and its malarial congeners or simulators. Especially difficult do I find it to distinguish between many *remittents* and *enteric fever*. For instance, one sees not unfrequently a continued type of fever, with great vital depression, and perhaps mental perturbation, which is uninfluenced by anti-periodic remedies, such as quinine or cinchona, or but to a slight extent—at any rate cannot in any sense of the word be *cured*, or rather *cut short*, by their use. Yet there is no eruption, no diarrhoea, &c. Say the patient dies; the chances are (for I have frequently observed this) that *no specific bowel or other typhoid lesions* are met with. If the case recovers the doubt still holds good, as one man will return it as "typhoid," another as "remittent," and yet of course neither diagnosis is absolutely reliable. It is because of the almost unrivalled opportunity I enjoy here of being able to confirm clinical observation by post-mortem examinations (or otherwise, *i.e.* of correcting it) that I still feel very sceptical as to any considerable prevalence of true enteric fever (a fever which, as I understand, has certain infallible anatomical signs discoverable post-mortem, whatever may be the variations in symptoms during life) among the natives of this country. Among Europeans (pure) this may be somewhat different. If I were asked to formulate an opinion upon this question, I would put it somewhat in this way :—

'1st. That enteric fever is a disease which undoubtedly prevails in India in both Europeans and natives, and has in both the same anatomical signs or lesions as the true disease in Europe.

'2nd. That it, however, probably prevails much more largely among Europeans than natives, and that young Europeans, newly arrived in the country, are most susceptible to the disease.

'3rd. That there are no absolutely distinctive signs or

symptoms by which enteric fever, as it occurs in this country, can be distinguished *during life* from certain continued or malarial fever, notably the so-called “adynamic remittent fever.”

‘4th. That it follows that many such cases, *i.e.* of enteric fever, are overlooked or wrongly classed as remittent or continued fevers, and *vice versa*’.

‘5th. That in not a few cases the etiology of the disease seems to differ from that usually assigned to it in Europe, viz. specific fæcal contamination, but may arise possibly from climatic causes, combined with non-specific fæcal evacuations or other like poisonous material productions the result of insanitary conditions in dwelling-houses, sewers, cesspools, drinking water, and all other sources of personal human contamination. And in support of this view it may be said that the disease in this country, especially among natives, is sporadic, not epidemic; it affects individuals rather than communities, and thus exhibits a behaviour quite different to that of the specific poison and its resulting phenomena in temperate regions or climates.

Dr. Alfred Clarke, of the Army Medical Department, writes:—‘The opinion of the staff is not entirely unanimous as to the etiology of enteric fever; some hold that a specific poison or germ is not absolutely a *sine quâ non*, but that ordinary filth-causes may develop it *de novo*; that climatic influences, acting on young and undeveloped constitutions predisposed in some way specially to develop typhoid, may also start the disease. The experience of our soldiers in Zululand seems to confirm this view. I feel sure I have seen genuine enteric fever in India, where all filth causes, in the ordinary sense of the term, were absent; and once started, the disease may spread rapidly, or appear to do so, without being actually contagious. The outbreak in Natal, January 1882, is an example.’

Dr. W. B. Beatson, Deputy Surgeon-General, Lahore, an officer of very large experience, writes:—‘I think that remittent fevers in India often imitate typhoid, and are described as such.’

Dr. Oldham writes from the Punjab, November 9, 1881:—‘There is a great deal of fever in India of continued type, of purely climatic origin, which so closely resembles enteric fever as to be distinguished from it with difficulty. Cases of this form of fever have doubtless been returned as typhoid. Since my attention has been drawn to this subject I have not had an opportunity of examining after death the bowels of a case of climatic continued or remittent fever.’

Dr. Whytlock, of the 38th Regiment, reporting on the state of health of the regiment at Peshawur in 1869, remarks:—‘Peshawur being certainly one of the most, if not the most, malarial and unhealthy occupied stations in all India, miasmatic disease is particularly prevalent at all seasons, but more especially about September and October. Ague then prevails to an enormous extent, the early and late cases assuming all the appearance and symptoms of cholera, and to the uninitiated being very alarming. There is every range of fever, from ordinary intermittent to *remittent and typhoid*.’

Dr. Woodward, of the United States Army, says:—¹ ‘The malarial influence and the pathological processes to which it gives rise are not merely manifested by the frequency of ordinary ague; it colours and complicates other diseases to an extent which can hardly be credited by those who have not been an eye-witness to its effects. In the fall and early winter of 1861 reports began to come from various quarters that a new form of fever was prevailing in our camps. The medical officers were well acquainted with ordinary typhoid, and it was precisely these men who first called attention to fevers that differed in many important particulars from those to which they were accustomed at home.’

A Board of Inquiry, after careful examination, recorded the opinion ‘that, while a certain number of cases of ordinary typhoid existed in the army, the large majority were bilious remittent, which, not having been controlled

¹ *Report on Typho-Malaria.*

in their primary stages, have assumed the adynamic type which is prevalent in typhoid fever. The cases, in great number, were studied by the best instructed medical men, who, recognising an unusual type, called it 'Chickahominy fever.'

Dr. Woodward, believing this form of fever to be the result of the combined influence of malarial poisoning and 'the cause of typhoid fever,' proposed the name typhomalarial, which was adopted. He says:—'This is no new thing. In every great army that ever yet campaigned for any length of time in a malarial region, the prevalent form of fever has been a hybrid between malarial fever and some form of typhus, the causes acting with peculiar intensity on strangers. The morbid conditions may be modified by a scorbutic taint.' He refers to many occasions on which this fever was observed, and notes especially the 'morbus mucosus' which occurred at Göttingen in 1760–61, where it seems to have alternated with intermittent, remittent, and dysentery. It was a fever which lasted 21 days, or sometimes 30 days; some cases proved fatal as early as the ninth day. It began as remittent or tertian, merging into the continued fever; during convalescence it reverted sometimes to the intermittent type. It had otherwise all the symptoms of typhoid; delirium, frequent feeble pulse, diarrhoea, meteorism, in the worst cases spots; tongue furred and swollen, with red papillæ protruding, dry and brown as the disease progressed; hæmorrhage from the nostrils about the sixth day; still more frequently hæmorrhages from the bowels. Peruvian bark proved highly efficacious in those cases in which the remissions were most marked.'

It received the name 'mucosus' from the belief that an excessive secretion of mucus from the alimentary canal was its most characteristic phenomenon. Röderer and Wagler described the morbid appearances found after death.

In one of the autopsies the agminated glands near the ileo-cæcal valve were marked with black pigment, the

'shaved beard' appearance, and the mesenteric glands were enlarged. Evidence of peritonitis was often present. Dysenteric sloughs were frequently found in the colon, but the bulky tumefaction and ulceration and sloughing of Peyer's glands is not recorded as having been present. Dr. Woodward says he is by no means sure that this essential lesion did not exist in some cases. Perhaps some of the gangrenous spots described as existing were of this nature. The Göttingen observers describe this epidemic as 'the corrupted and degenerate progeny of intermittent fever,' and they thought they saw also a causal relationship between intermittent fever and dysentery, an opinion which Dr. Woodward says that he shares with them, and so do I; for the more one studies these fevers and dysentery as seen in India, the more closely do their etiological relations seem to be drawn together.

The Walcheren diseases are referred to in the same connection. They were diarrhoea, dysentery, intermittents, and a form of fever which began as a remittent, and subsequently assumed a continued form, which at that time was designated the Walcheren remittent. Davis tells us that, beginning as an intermittent, the most common form being the double tertian, it assumed a continued typhoid type as it progressed, with muttering delirium, small rapid pulse, dry, black tongue, sordes on the teeth, and fetid and black discharges from the bowels.

Dr. Drake has shown that in a general way these fevers are most intense on the border of the Gulf of Mexico, and gradually diminish in frequency and severity as we go to the north, no longer prevailing in an epidemic form beyond the forty-fourth parallel, and ceasing to occur altogether at about the forty-seventh. He says:— 'I need only remind you of the frequent occurrence throughout the Southern States, side by side with the ordinary agues, of malignant forms, the so-called congestial chills or pernicious fever, of the severity of remittents which prevail, of the frequency of big spleens, disordered livers, and malarial anæmias. Next, let me remind you of

the important fact that intermitting and remittent fevers often disappear, more or less completely, from a neighbourhood in which they have long prevailed, and are replaced by typhoid fever.' Drake calls it 'properly an autumnal fever, and is an equivalent for the true remittent of the warmer climates. Again, the substantial truth is that in numerous districts intermittents and remittents were the prevailing form of fever when the first settlements were made; that as time passed by and cultivation progressed, the intermittents diminished in frequency, the remittents exhibited more and more a disposition to pass into continued forms, and finally were replaced by ordinary typhoid.'

Professor Dickson says:—'On examination typhoid lesions will be sometimes found in the body dead of bilious remittent. The mucous membrane of the stomach and intestines is highly injected in severe and short attacks. In more protracted cases follicular ulceration may be found throughout the whole extent of the bowel.'¹

Speaking of post-mortem examination of persons dying of this fever, Dr. Woodward says:—'That which has been emphasised by Röderer, Wagler, and Dickson between the simple inflammatory enlargement of the closed glands and the more luxuriant process which occurs in typhoid, every possible transition existed. I for one confess myself unable to draw a line between the two conditions. . . . The sloughing and ulceration is, I think, sufficiently well explained by the intensity of the process and the nutritive disturbances which then result, without conjuring up in our imagination an undemonstrated specific something to account for it.'

It seems tolerably clear from this that in America the existence of climatic fever with ulceration in the small intestines, distinct from the specific enteric fever, is recognised; it has been placed apart, and is regarded as the result of the combined action of a malarial and typhogenic poison, though there are not wanting indications that it

¹ 'On the Blending and Conversion of Types in Fever.' By S. H. Dickson, M D. *Trans. American Med. Association*, vol. v., 1852, p. 127.

may be the result of progressive action of a febrile condition, however set up.

Turning to another quarter, I find the view of typhoid fever of a non-specific character strongly advocated by Professor Léon Colin, of the Vâl-de-Grace, a medical officer of eminence in the French army, whose experience and researches on the subject of malarial and typhoid fevers give great weight to his opinion.

He says:—‘A theory was advanced by M. Boudin that intermittent and typhoid fevers were antagonistic, and that where one existed the other was absent; but it has been abundantly shown that not only in Algeria but in Italy the mortality from both has been excessive. In Algeria, 4·63 per 1,000 men died of typhoid; 3·05 in France itself. In Algeria it caused as much mortality as intermittent. In Rome the French army lost in 1868 the enormous number of 203 per 1,000 from typhoid alone. These facts prove that malaria confers no immunity; indeed, intermittents and typhoid appeared simultaneously in the same regiments in Algeria and in Rome.’

In India localities notoriously malarious are not specially remarkable for the prevalence of typhoid, but it must be remembered that no part of India, except the hill stations, can be regarded as exempt from malarial influences; and that no station where Europeans are located is exempt from typhoid. There appears to be nothing in India to support the theory of antagonism between malarial and typhoid fever; but if, as is thought by some, fever with enteric ulceration is of miasmatic origin, the question of relative prevalence and mortality of enteric and paroxysmal fever proves nothing more than that, under the circumstances, the fever had assumed one type rather than the other. And it is manifest that very careful and close analysis of the history of individual cases and outbreaks in Europeans and natives should be made, especially in such developments of fever as have occurred in regions like Burdwan, the Doab, and other districts where low and continuous forms of fever, ascribed

to miasmatic influences arising from water-logged land and organic decomposition, have prevailed. Some interesting reports have been published on the Burdwan fever, and I would notice especially those by Drs. French and Roy, of the Bengal Service; they attribute it, as do others, to paludal influences, and in their account of the cases that assumed the low remittent type there is much that is suggestive of fever attended with enteric complications.

Dr. Jackson¹ describes it as follows:—‘In every locality visited by me I found existing—(1) a multitude of chronic cases, of which the type was almost always intermittent, very rarely remittent. In very recent cases the intermission was quotidian, in the older cases quartan or quintan; (2) a small number of acute cases of continued fever, with no well-marked remission whatever; no greater amount of remission, in fact, than accompanies all cases of acknowledged continued fever. In these cases there was no premonitory ague or cold stage; after two or three days of lassitude, loss of appetite and malaise, the fever appeared, the skin becoming hot, the head heavy, the tongue coated with grey fur, the urine scanty, and the bowels confined. Sometimes nausea was present, but oftener not. There was always distaste for food, perverted taste, and thirst. From the very first the patient had a stupid, drowsy, brain-poisoned aspect, and was unwilling to answer questions; the intellect gradually became more and more confused, and he lost the power of understanding what was said to him, and it was only by shouting that he could be roused at all. From the first also there was intense prostration and loss of muscular power. In the cases that proved fatal the stupor passed rapidly into coma, and after twelve or thirty-six hours of utter insensibility death occurred. Some of these cases proved fatal in three days, others lasted a week or ten days. In the more protracted cases which proved fatal there was lung engorgement and pneumonia, the urine and fæces were often passed involuntarily. The most striking symptom was the early stupor, and the rapidity with which coma supervened. In such cases the conjunctivæ often remained perfectly clear and uninjected to the last. Hepatic or splenic tenderness or enlargement was rare. Acute delirium was not very common, but I was told by villagers of cases in

¹ *Report on Burdwan Fever.*

which all those which proved fatal were accompanied by delirium, the patients endeavouring to run out of the house to drown themselves. These acute cases are numerous enough, they constitute *the* fever, but my visits to the infected tracts were made in December, January, February, March, and April, when matters have begun to mend, and though such cases are met with up to March, they are comparatively few in number after December.'

He further writes:—'As regards the fever, the acute cases, which the natives call *jwor-bekar* and *mootun-jwor*, it reminded me very much of typhus. I sometimes looked for, but never succeeded in finding, an eruption. I never saw any case which led me to connect it with enteric fever. One very noticeable circumstance about the disease was the horrible sickening odour exhaled by the sick.

'Quinine, which was very efficient in chronic intermittent cases, had no effect on the acute cases, and failed entirely in my hand to prevent a relapse. I had no opportunity of making any careful clinical study of the diseases. I never saw any one case from the beginning to the end. I have seen the same case but half a-dozen times during the course of the original attack, period of freedom, and relapse. I have seen cases during every stage of the disease, and been present at very many deaths. The first indication of the presence of the fever in a village is the occurrence of a few cases of this *jwor-bekar*, of which comparatively little is thought at the time; in the ensuing year, however, more of these cases occur earlier in the season than the ordinary malarious fever, and multiply with great rapidity as the year advances. I have noticed that where deaths from this cause occur during the year of invasion, there are deaths in *the same houses* during the following year in more than half the number of cases noted.

'A son would tell me that his father had been out of sorts and oppressed for two or three days, and that while they were out in the fields together he had suddenly declared that he was unable to work any more and returned home. He had no fever, no attack of shivering succeeded it, but he became hot, ached all over, was sullen, could not eat, did not like people to talk to him, only spoke to ask for water, and was heavy and apathetic. He was never very hot, but the fever never went away; sometimes he would groan, but was generally quiet. The bowels were confined. He still would not eat, and became angry

when pressed, or if spoken to; then he seemed stupid, and he could not understand what was said to him. Sometimes he had to be called many times before he could be roused; then he became insensible but could breathe. Well, afterwards he became like this. He had been ill three or four days, sometimes a week. There was no enlargement of the liver or spleen. I could find no eruption, the man was comatose, had hypostatic pneumonia, and was dying.'

I cannot help thinking that examination of the intestines might have discovered enteric ulceration, whether precisely similar to that of specific typhoid I cannot say. Unfortunately there are no autopsies recorded, and this is one of the great difficulties attending study of disease in India. That typhoid fever with ulceration occurs in India among the native population as it does among the Europeans is beyond dispute (as you may see in these drawings); but how much of it depends on specific poisoning; how much on general causes arising from heat, miasmata of vegetable or animal decomposition, or of both combined; and what are the distinctive phenomena in life and anatomical lesions after death, are subjects that require further inquiry. I have no desire to dogmatise on this subject, but I would repeat my conviction that there is much fever of climatic origin which is as like specific typhoid as one case of typhoid may be like another, and that it is of the same character as that called by Americans 'typho-malarial,' and by the French 'typhoïde palustre.' I am aware that this opinion is not accepted by all, but, after many years' experience, such is the conclusion I have arrived at, and I find that similar views are entertained by others. I do not say that this form of fever differs in its pathology so much as in its etiology. It seems to me quite possible, and my presumption is supported by experience, that though a disease, of which the cause has never been actually demonstrated, has been logically traced to a specific origin in temperate climates, it may have other sources of development in India.

To return to M. Léon Colin. He says that authors of great weight have expressed the opinion that paludal typhoid is the result of the combined action of paludal and typhoid elements, and that the compound name of the fever perfectly indicates the composite nature of its cause. This condition he maintains may occur; he has recorded cases of it, and refers to some that occurred at Nancy in 1875, when the infection was at the same time telluric and putrid. Other examples were observed in an epidemic at Avranches in 1873. But otherwise the fever is the transformation of paludal into typhoid fever, and he is of opinion that all acute febrile conditions, accompanied by a marked alteration in the secretions and by gastro-intestinal complications, may induce the spontaneous development of typhoid,¹ and that in such cases it is natural that it should be impossible to recognise the affection during life, for the two diseases have ceased to be distinct, the remittent fever being transformed into typhoid.

He gives numerous examples and post-mortem examinations in support of his views. One is as follows: M. Maillot found, in the case of a person who died of pernicious remittent, the small intestine was normal to within two feet of the ilio-cæcal valve, where ulcerations to the number of twenty-five or thirty were found.

Nepple also gives a similar case, in which there were ulcers in the jejunum and ilium of the size of a centime, with gangrenous surfaces on the elevated patches of ulceration. The mesenteric glands were white and hard, and of the size of a nut (*noisette*).

Linguette, in Cochin China, has also shown typhoid complications of pernicious fevers, like those which occurred in Algeria and in Rome.

M. Colin refers to the opinion expressed at the Academy of Medicine on the malarial origin of typhoid fever, which does not admit of the antagonism supposed to exist

¹ 'De la Fièvre Typhoïde Palustre,' *Archives Générales*, 1878, t. i., p. 276.

between malarial and typhoid, against which so many proofs exist. It allows that paludal influences may confer a character (*cachet*) on epidemics, but cannot originate typhoid; and that when it appears under these circumstances the origin must be sought in the general state of hygiene of the towns or houses where the endemic tendency prevails. M. Colin says that hygienic conditions must be known before we attribute the fever to marsh exhalations, and that he also is of opinion that animal miasmata, above all others, are concerned in the production of this disease. He gives further instances of other epidemics observed in France, especially by Gaultier de Claubry at Carentan (Manche), and by M. Gintrac, at Sainte-Croix de Mont-Carentan, near Bordeaux, in which typhoid occurred as a result of malarial poisoning, and I would refer you to his remarks in these reports in the 'Archives Générales.'¹

' D'après la manière dont nous comprenons la genèse de la fièvre typhoïde palustre, qui n'est pour nous qu'une transformation d'une fièvre à quinquina, il n'y a point à demander au sol la raison directe de son développement, dont le malade lui-même est l'intermédiaire; c'est par l'altération de l'organisme que, primitivement palustre, la maladie devient secondairement typhoïde, trouvant dans cette altération l'élément typhoïgène qui manquait pour sa formation d'emblée.

' Plus on avance vers le Sud, plus les manifestations de l'impaludisme chez le nouveau venu tendent à la continuité et à l'intensité du mouvement fébrile; n'est-ce pas là une des raisons de la prédominance habituelle de la fièvre typhoïde dans les garnisons du sud-est de la France? En des régions plus septentrionales, la transformation typhoïde des accidents palustres ne s'accomplira qu'en des années exceptionnellement chaudes; comme l'a été l'année 1874. C'est en ces conditions seulement que les manifestations de l'impaludisme acquerront l'intensité voulue pour cette transformation.

* * * * *

' En résumé, le groupe des fièvres typhiques, et spécialement des fièvres typhoïdes, est séparé de celui des fièvres palustres d'une

¹ *Loc. cit.* pp. 436-441.

façon moins absolue qu'on l'admet en général. Des faits nombreux démontrent le mal-fondé de l'intolérance dogmatique qui, si souvent, a fait récuser d'autorité les observations des auteurs qui ont démontré ce rapprochement.

' Si les points d'appel ordinaire de la fièvre typhoïde sont les centres peuplés où prédominent les miasmes d'origine animale, l'affection peut aussi résulter des émanations telluriques. Elle en provient, suivant nous, d'une manière indirecte, grâce aux modifications subies par l'organisme, sous l'influence des formes les plus fébriles de l'impaludisme, spécialement des formes continues et rémittentes. Le miasme palustre provoque, pour ainsi dire, le miasme humain qui intervient, ici encore, comme facteur indispensable de la fièvre typhoïde. La maladie, dite fièvre typhoïde palustre, est dans la majorité des cas, non plus le résultat pathologique de l'association primordiale de deux éléments, l'un palustre, l'autre typhoïgène, provenant du milieu ambiant ; elle résulte de la transformation, dans l'organisme lui-même, d'une forme primitivement palustre.

' Elle fournit la preuve de la spontanéité de la fièvre typhoïde.

' Si elle est rare néanmoins, dans les régions à malaria, c'est que son apparition est habituellement subordonnée aux deux circonstances suivantes :—

1° Elévation considérable, climatique ou saisonnière, de la température.

2° Arrivée récente de l'individu dans un foyer palustre ;

' Circonstances qui, d'ailleurs, constituent elles-mêmes les conditions d'explosion des formes les plus intenses, au point de vue fébrile, de l'impaludisme (fièvre rémittente d'été, ou subcontinue estivale).'

On this subject Professor Colin has written to me as follows :—

' Mes recherches personnelles m'ont inspiré la conviction que la fièvre typhoïde est, et a toujours été, fort commune dans les pays chauds ; qu'elle y a été longtemps méconnue, parce que l'on ignorait ses symptômes et ses lésions ; que sa fréquence y a été grande, surtout quand on a envoyé en ces pays des contingents considérables de jeunes soldats.

' Je donne la preuve de ces faits à la page 31 d'une brochure que j'ai l'honneur de vous adresser sur la *Fièvre typhoïde palustre*.

‘Quant à la nature et à l’origine de cette fièvre typhoïde des climats chauds, elle peut certainement dépendre de l’influence des foyers *typhoïdés* analogues à ceux des climats tempérés (miasmes des égouts, des matières fécales, etc.) ; mais, suivant moi, elle tient surtout à la transformation, dans l’individu lui-même, de la fièvre malarienne qui infecte l’organisme, et met celui-ci en puissance de créer la fièvre typhoïde par auto-infection.

‘Dans cette même brochure j’indique pourquoi cette transformation a lieu surtout chez les individus jeunes, nouvellement expatriés, et dans les pays chauds où l’empoisonnement malarien donne lieu à des accidents fébriles spécialement intenses.’

Dr. J. Wise, late civil surgeon of the large and important district of Dacca, in Eastern Bengal, has studied the fevers of that part of India with great care for many years, and had great opportunities of observing and recording all that is connected with their etiology, symptomatology, and pathology. He has noted the occurrence of bowel complications in malarial fevers, and has recorded¹ the cases in which ulceration has been found, viz. :—

Muco-enteritis, extending from the stomach to the colon, with dysenteric ulceration.

Remittent fever with enteric symptoms.

Malarial pneumonia with ulceration of small intestines.

Tubercular disease with ulceration of intestines.

And after a long and closely-reasoned description of the symptoms of remittent and enteric fever, and a careful comparison of all the points in which they resemble or differ from each other, and from true enteric fever in Europe, he records his opinion on the etiology of the enteric fever of the natives of Bengal :—

‘My own opinion, after much consideration and observation, is that it is a true paludal or malarious fever, due to the same exciting causes as are universally believed to produce ague, namely, to miasmata given off from decaying or fermenting organic matter, and not necessarily

¹ In an inedited MS. of a work on Fever.

connected with emanations from drains, cesspools, or privies.

‘In expressing thus unreservedly an opinion that enteric fever may be of malarious origin, those who disbelieve in the existence of such an element as malaria will marshal arguments which may appear directly opposed to what is here stated. The irresistible mass of facts collected by Murchison, Budd, and others, prove that enteric fever in Europe is truly pythogenic, and that in most instances it is caused by decomposing feculent matter. In India we cannot accept that as the sole, or perhaps chief, exciting cause of enteric fever. In asserting its malarious origin no new or startling theory is advanced. Similar opinions have been ably maintained by many European writers, and by none more frankly than by Dr. John Harley.’¹

He recognises three varieties of enteric fever: ‘the simple inflammatory, the contagious, and the paludal.’ The last he believes to be the most common. It is non-contagious, its course is usually slow, and it arises from putrescent animal and vegetable substances.

‘In the last edition’² of Dr. Murchison’s great work there is nothing antagonistic to the conclusion arrived at by Dr. Harley; rather, there is much to confirm it. Referring to the antagonism supposed to exist between enteric fever and the common paroxysmal fevers, Dr. Murchison, on the authority of M. Boudin, quotes several remarkable instances in which French regiments on their return from Algiers remained exempt from enteric fever which was prevalent among other soldiers residing in the same barracks. But, as Dr. Murchison shrewdly remarks, this and other similar facts mentioned suggest a similarity instead of an antagonism. By instances drawn from malarious countries, such as Lorraine and Bohemia, he verifies this conjecture, and shows by two striking

¹ Reynolds’ *System of Medicine*, vol. i., pp. 601–608.

² *A Treatise on the Continued Fevers of Great Britain*, second edition, 1873, p. 451.

examples that the identity of the exciting causes in both diseases is not only exactly the same, but that they are generated under the same conditions.¹

‘Dr. Parkes² also entertains doubts that the generally accepted cause is the only one to which enteric fever is to be referred.

‘No one, however, has more persistently and ably affirmed that enteric fever is generated in India by other causes than fæcal emanations than Dr. Bryden. In 1872 he wrote :—“ Eight years since, from the facts then at my disposal, I made the generalisation, that the typhoid fever of the British soldier in India is primarily due to climatic influences. The belief that defective conservancy will be found in every case when typhoid fever shows itself is very apt to lead to the conclusion that any statement to the contrary must be erroneous. This is a narrow view, and it is not warranted by any feature in the aspect of typhoid as we meet with it among our soldiers.”³

‘There are several points which conclusively indicate the presence of some exciting cause distinct from and independent of any fæcal emanations.

‘The Dacca gaol and lunatic asylum, in which observations were chiefly made, stand side by side on the most elevated, and consequently driest, piece of ground within the city. The land falls away to the north and south. No water lodges, and no sewers exist in either institution. The dry-earth system of sewage is followed, and the whole of the night soil is buried in the gardens, and vegetables planted as soon as the pits are filled in. In both places the gardens lie on the north-west of the dormitories, and the wind during the hot and sickly months blows steadily from the south-west.

‘The drinking water supplied to the prisoners is derived from a well to the south-east of the wards. It was analysed in 1871, and pronounced to be purer than the generality of wells in Dacca.

¹ *Op. cit.*, p. 494.

Practical Hygiene, ed. 1866, p. 455.

² Appendix to the ninth Report of the Sanitary Commissioner with the Government of India, 1872.

‘The lunatics, again, obtain their drinking water from the river, whence it is brought in carts, and then filtered through charcoal and sand before being issued.

‘The fact that no outbreak of enteric fever has as yet occurred in either institution, and that only isolated cases are met with at intervals of weeks or months, seems to refute the idea that the seizures were due to any local defects within the walls.

‘If enteric fever were in India, as it undoubtedly is in Europe, due to faecal emanations, how can we explain the circumstance that in the midst of the city, containing, as it did in 1872, 69,000 inhabitants, densely populated quarters are to be seen in which the faecal deposits of generations are collected in unsightly heaps, or thrown into privy wells within a few feet of the well from which drinking water is obtained, which causes diarrhoea when first used, but never any form of fever is observed? Toleration, however, is soon established, and comparative health enjoyed. With the exception of one case in 1874, not a single person was admitted into the Mitford or Public Hospital with enteric fever between November 1866 and November 1874.’

The following remarks by Dr. Wise on malarial pneumonia with enteric symptoms are interesting and valuable : ‘The disease with which paludal enteric fever is most frequently and easily mistaken is asthenic, or typhoid, pneumonia. This is a most common disease in Bengal, either appearing idiopathically, or secondarily, to other diseases. Among the natives it generally assumes the type called bilious, in which gastric and enteric symptoms are most conspicuous.

‘In thirteen cases I made careful examination of the small intestines after death from this disease. In ten, congestion, more or less intense, and varying from a pink to a port wine colour, with patches of extravasation, were found in the mucous membrane of the ileum; in one an ulcer of the duodenum, with extravasated spots in the ileum, existed; in one Peyer’s glands were congested and pitted, while the mucous membrane around was deeply

injected; and in one Peyer's patches were singularly distinct, their surfaces being humid and reticulated, the solitary glands were swollen and mammillated, and the mesenteric glands were enlarged, containing a milky fluid like chyle.

'But pneumonia with typhoid symptoms is no less common as a complication of intermittent or remittent fever—the febrile pneumonia of Morehead. As cases are seldom seen among natives until at least a week has elapsed, it is often difficult to distinguish the primary disease. If the patient is intelligent, and is seen early, a correct conclusion may generally be arrived at; but when he is delirious, with a black furred tongue, has twitching of the muscles and diarrhoea, it is often impossible to decide.

'Pneumonia with enteric symptoms is often, I believe, confounded with pneumonia secondary to paludal enteric fever. In my experience, inflammation of the lungs rarely appears in the course of any of the malarious group of fevers before the end of a week, while in enteric fever it is generally during the third week.

'In all cases of doubt, twenty-grain doses of quinine given twice a day, or five-grain doses with antimony every four hours, as recommended by Morehead, will decide the question whether the disease is secondary or not.

'There is nothing more certain in medicine than this. A few doses of quinine given to a patient prostrate with "febrile pneumonia" works a wondrous change; they check the fever, and the patient passes in a few hours from a state of misery into one of comparative ease and health. Of all the practical benefits conferred by Dr. Morehead on the people of India, none probably will be more enduring than this one, which he was the first to recognise.'

I am indebted to Surgeon-Major A. Clark for the following note:—

'Typhoid fever has prevailed very extensively in Natal and Zululand during the war. Since January 1, 1879, to May 31, no

less than 267 admissions for enteric fever have been recorded. Many of these occurred in healthy camps on ground previously unoccupied (virgin soil), and in bodies of picked men. The water supply, as a rule, good; no sewers or drains; conservancy, dry earth or trench, and carefully attended to. The troops of Crealock's Division, which were encamped near the coast and in close proximity to marshy ground, where the natives suffer severely from remittent fevers, had more admissions from enteric than the Second Division, which was inland and generally at higher elevations. The medical officers differed as to this fever, several maintaining it was not typhoid as commonly understood in England, but bilious remittent with typhoid symptoms, as seen in India; others nailed their colours to the mast that it was genuine enteric. In support of the former, a medical officer just home reports that numerous cases entered as enteric were discharged, and at their duty five or six days afterwards. Surgeon-General Woolfreyes describes this fever as "typho-malarial." He says:—"It is not a fatal fever, but it causes great prostration, and a change to England is absolutely necessary. I am of opinion that it is climatic, the true autumnal fever. It, as a rule, commences with sore-throat, a peculiarity; the rose spots are invariably present, and in fatal cases the lesions of Peyer's patches are well marked." At the time that enteric was reported so common in the First Division, jaundice also prevailed very extensively, but was unknown in the Second Division. Enteric has also been reported as causing much sickness amongst the troops in Afghanistan. Here, again, camps were often pitched on virgin soil, though the water supply was far from satisfactory, and dead camels so constantly polluted the streams. It prevailed *with* cholera and severe remittent fevers. Altitude made no difference, cases being admitted in camps several thousand feet above sea-level. Enteric cases are reported from nearly every station in the Bengal Presidency, some such as Cambellpore, in the Punjab, where the 'filth' element is at a minimum. At this station there is no large bazaar or city in the vicinity; the soil is arid, sandy, and very dry; the rainfall exceedingly small; water-supply good, and very carefully filtered and attended to; the dry-earth conservancy is carried out to perfection; the milk danger is almost *nil*; yet enteric occurs, and amongst the troops who drink hardly any milk, while the women and children, who probably drink a good deal of bazaar milk, have so far escaped. These facts seem

to show that enteric fever cannot be always ascribed to a "filth" cause, but that climate, or what is embraced in that unknown word "malaria," may give rise to it, as it undoubtedly does to intermittents, remittents, and *possibly* cholera.'

Climatic fevers of a continued or continuo-remittent type appear to have been observed by several medical officers besides those I have mentioned.

Dr. Hoystead, writing from Hyderabad, Scind, speaks of 'the close affinity which exists out here between pernicious remittent fever and typhoid'; and Staff-Surgeon Maclean, R.N., writing from the Royal Naval Hospital, Ascension, March 1881, alludes to the occurrence of enteric fever in a locality, and under circumstances of close observation, where no connection could be traced with defective sanitary arrangements, though it is probable that malarial influences do occur.

Staff-Surgeon G. Maclean, R.N., says:—'I think my experience of enteric fever as it has prevailed in this isolated spot since my arrival in June 1878 may be considered of sufficient interest to excuse my addressing you on the subject. During this period cases of the disease have occurred from time to time, without any discoverable connection with each other, and in the great majority of cases in the persons of new comers. In every instance the disease was closely marked by the usual characters, and could not possibly be mistaken for any other. If enteric fever, according to the commonly-accepted theory of its origin, is always associated with defective sanitary arrangements, then Ascension, of all places with which I am acquainted, ought to be exempt from it. There is no such thing as a sewage drain or cesspool in the island, all sewage and other filth being removed daily and thrown into the sea, to leeward of all dwelling-houses. The water (partly collected from the roofs of buildings during rain, and partly condensed) is stored in iron and cemented stone tanks removed from all possible source of contamination, with the exception of the summit of Green Mountain, where there is a soil resulting from the decomposition of grey trachyte (the fundamental rock of the island),

giving rise to a feeble vegetation. The island is a mass of volcanic rock and ashes, incapable of supporting any vegetable life whatever.

‘My investigations have utterly failed to connect the fever with any of the conditions commonly believed to be essential to its production. The idea of a climatic origin did indeed occur to me; but it seemed like heresy to suggest such a thought, and I was obliged to be contented with the fact of the existence of the disease without pretending to explain its origin. I am encouraged to send this communication, in the hope that it may in some degree contribute to the establishment of the view that under certain as yet unknown meteorological conditions a disease indistinguishable in its symptoms and pathology from enteric fever may arise independently of a specific cause.’

Dr. Don, of the Army Medical Department, has expressed his views on the subject of the climatic origin of fever with enteric complications. He says: ‘—Doubts are now pretty freely expressed on the causation of the disease, especially by medical officers, whose experience of it extends to all parts of the world. In the summer of 1878 I saw about a dozen cases in Gibraltar, all of which presented the characteristic symptomatology of enteric fever, just as we might see it in London, and examination of the fatal cases revealed the essential bowel lesions. During a three years’ tour of service in Bermuda, in charge of the Royal Engineers, I saw many enteric fever cases in my own corps and in the regiments quartered in the island. But in a large proportion of these cases the symptoms were not altogether pathognomonic; for instance, I cannot honestly say I ever saw the characteristic rash; nor were the stools ochry, but nearly always green, dark, or bilious. Some may say these were not cases of true enteric fever; but I answer, if not, what were they? For in the fatal cases the essential bowel lesion was found; nay, some even died of perforation.

¹ *Transactions of the Epidemiological Society*, vol. iv. p. 299.

‘The early symptoms of these fevers are usually so much alike, that it is often quite impossible on admission to determine what form may ultimately supervene. Many times have I seen cases admitted under febricula, then change to simple continued, and finally to enteric, as the fever developed. This arose from no slovenly diagnosis in the first instance, but was forced on us by the super-vention of phenomena which it was quite impossible to anticipate. They occurred simultaneously, concurrently, and mixed up at the same time and place, in the same regiment or community. From the same barrack or company room one man may be admitted into hospital with febricula, a second with simple continued, and a third with enteric, all on the same day. All these fevers have the same seasonal period of prevalence. During winter and spring they are at a minimum, or wholly absent; from June to October is the season of their prevalence north of the line, with a corresponding reverse period in the southern hemisphere.

‘Now when such allied forms of disease as these fevers present such points in common, may we not reasonably infer that their causation must have also much in common? If the exciting causes are quite distinct, who can differentiate them? If subjection to apparently identical influences results in the evolution of three different forms of fever, what explanation have we to offer? My impression is that with varying degrees of intensity the exciting causes of all these forms of fever are, if not identical, entirely similar. I believe that the chief factors of these endemic fevers are to us intangible and purely climatorial. Local insanitation greatly aggravates and intensifies the type of the fevers, but it is not the main originating cause. If the general causation of these fevers is so vague, indefinite, and non-specific, on what principle then can their different forms be explained? Now, I doubt if such questions can be answered by reference to supposed exciting or external causes. 1

think they can be better explained by a study of the internal or predisposing causes :—

‘ 1. Constitutional, hereditary, or acquired predisposition.

‘ 2. Age.

‘ 3. Want of acclimatisation.

‘ I cannot help thinking that this enteric lesion is to be found in several forms of a closely allied fever, at the same time it does not necessarily imply a specific causation under every circumstances and in all parts of the world. It is only on some such assumption that we reconcile the mass of conflicting testimony and experiences which have of late years accumulated from all parts of the empire. We know the theories of Budd and Murchison as regards disease in this country ; and I think we must conclude with them, that in whatever mode it originates, when once set going it tends to multiply and propagate itself in directions suitable for its spread.

‘ Propagationists may say that the specific germs are always and everywhere present, and ready to develop under favourable conditions of temperature. Can it be logically maintained that these germs exist not only in every station in India, and in such places as Malta, or Bermuda, or Gibraltar, but even confront us when we penetrate into the virgin wilds of Zululand ? For in the late Zulu war cases of enteric fever occurred in detachments of our young soldiers, which no theory of specific contagion could explain, and which were usually vaguely accounted for on the pythogenic theory of foul drinking-water. In short, I think there can be no doubt that enteric fever cases constantly crop up in hot climates, the causation of which is inexplicable, either by a theory of propagation or of pure pythogenesis.

‘ In support of these views let me mention one or two instances which have come under my own observation :—

‘ The 15th Regiment landed at Bermuda, in a healthy condi-

tion, from New Brunswick, just at the beginning of the hot weather of 1868. It was, of course, a mistake to land them on a tropical island at the beginning of summer, and in six weeks' time this began to be painfully apparent. They were split up in detachments here and there on wild spots in Bermuda making a military road.

'First diarrhoea and mild dysentery showed itself, and, almost simultaneously, febricula, simple continued, and many cases of enteric fever occurred. Meanwhile the other troops in the island, who were more or less acclimated, had only the usual and moderate amount of summer endemic fever amongst them.

'The detachment of the 15th which suffered most—especially from enteric—was encamped on perfectly virgin sand, on the sea-shore, far removed from habitations, or even the very suspicion of faecal contamination.

'As regards food and water, although not shelter, they were on a par with other troops who did not suffer from enteric fever, except in the usual sporadic way. In fact, there was nothing to account for the fever, except the circumstance that the men were brought to Bermuda at the wrong season of the year, and perhaps imprudently placed under canvas; in short, the cause was nothing special, but a general climatorial one

'The principal medical officer ascribed the sickness of the regiment to "the effect of a sudden change from the cold bracing climate of New Brunswick to the hot relaxing climate of Bermuda." The surgeon of the regiment, referring to the prevalence of enteric fever in another detachment, said he "found it impossible to lay his finger on any specific cause for the prevalence of this fever." I can well endorse this statement, for I was in Bermuda at the time, and know how carefully the possible and probable causes of the fever were looked into.

'Over and over again, in Bermuda and Gibraltar, I have seen isolated cases of enteric fever, both in barracks and private houses, which a filth theory could not logically explain; for if such cases arose from soil or water pollution, how came it that scores of other people living under identically the same conditions were not similarly affected? Or, if soil and water contamination is the cause of the disease, why should it be so only in summer and autumn? Such filth-sources of fever exist equally all the year round; as much in the mild temperate winter and spring as in the hot summer. If a cesspit system, and a porous

absorbent rock, be at the bottom of the mischief in Bermuda, such cannot be in Gibraltar, where there is impervious limestone and elaborate main drainage and sewage conservancy. And so, by a process of logical involution, the etiology of enteric fever in the tropics becomes a subject of exceeding difficulty and complexity. If we wish to arrive at the whole truth of the matter we are bound to look at causation from every possible point of view. This is no easy matter when preconceived opinions are apt to beset us on every side.' ¹

Surgeon-Major Martin, of the A.M.D., says: ²—'It would appear that the phase of enteric fever with which we are familiar in tropical regions teaches that the disease is to be considered as being in its nature more analogous, and in its history more closely allied to disease of climatic origin (using the term in a wide sense), than to disease of a specific nature.'

1. Enteric fever is produced by at least two causes: the virus of decomposing animal matter, and the specific virus of contagion.

2. Enteric fever may be in some cases, but certainly not in all, the result of a specific virus; infection from decomposing animal matter may produce enteric fever, and at the same time other affections as well as enteric fever. Specificity of a disease does not necessarily imply specificity of its cause. When putrefactive impregnation results in enteric fever it is certain conditions of the organism which determine this result. Besides the pythogenetic, I believe the determining conditions are—

1. Hepatic insufficiency.

2. An abnormal activity of the intestinal glands, consequent on and vicarious or supplemental to this hepatic insufficiency.

3. An idiosyncrasial proneness of these glands to assume their abnormal function.

Professor Bouchard ³ says that typhoid is a specific miasmatic fever, and that the *materies morbi*, not necessarily arising from a previously infected organism, may impregnate the air, soil, or water, and may be dissemi-

¹ Epidemiological Society's *Transactions*, 1881.

² *Contributions to Military and State Medicine*, p. 107.

³ *Revue Mensuelle de Médecine et Chirurgie*, Novembre 1877.

nated by man, by air, water, and other objects; but he considers that the doctrine of infection and contagion is too exclusive, that the doctrine of fæcal origin is too limited, and that of spontaneity is not proved.

I am indebted to Dr. Kynsey, P.M.O. of Ceylon, for the following interesting statement on the fevers of that island, which seem to be of a milder type than those of India. It singularly illustrates the confusion which still obtains about the nosology of tropical fevers, and attests the existence of a form of climatic continued fever which resembles the specific enteric fever that no doubt exists in Ceylon as it does in India.

‘The following table gives an abstract of the deaths during ten years according to medical returns:—

Fevers					Treated	Died	
Enteric	276	78	
Continued	2,539	149	
Febricula	617	1	
Ague	91,413	351	
Remittent	3,409	252	
					98,254	831	8 4 per 1,000

‘Death-rate from all causes in 1880 was 18·33 per 1,000.

‘Registrar-General’s returns for the island of Ceylon for 1880 :—

‘Deaths from all causes 50,575, or 18·33 per 1,000.

Enteric fever	5,005
Simple continued fever	5,779
Ague	22
Remittent fever	28

Total . 10,834

‘The population of Ceylon, which may be compared to Ireland in extent, was 2,405,576 in 1871 and 2,758,529 in 1881, showing an increase of 352,953 in the ten years.

‘During this period 98,254 cases of fever were regis-

tered in the seven provinces, with 831 deaths, a death-rate from fevers of 8·4 per 1,000.

‘Table I. gives in detail the varieties of fever with the mortality for each of the ten years of the period 1871 to 1880 inclusive.

‘Table II. shows the total island mortality, according to the Registrar-General for 1880, with the deaths from fever. The death-rate from all causes per 1,000 was 18·33. As regards the diagnosis of the causes of death it must be remembered that we have to trust to ignorant reporters. The diagnosis of the special kind of fever cannot be depended upon, but the total number of deaths from fevers is perhaps fairly correct.

‘There can be no doubt as to the existence in Ceylon of genuine enteric fever, but there is a strong tendency to call all protracted fevers by this name; a proof of this will be found by examining the tables annexed. In Table I. only 276 cases with 78 deaths of enteric fever were registered for the ten years in the medical report, whereas according to Table II. 5,005 deaths are attributed to this cause for 1880 alone. Enteric fever is confined to the large towns, occurs only in sporadic cases, and is never epidemic. The cause is, as far as my judgment goes, a specific poison; but I am convinced there is a form of fever in the tropics indistinguishable during life from this fever, and without the characteristic lesions of Peyer’s glands after death.

‘2,539 *cases* of continued fevers were registered in the ten years under review, and 5,779 *deaths* are alleged as due to it in 1880, showing again the untrustworthy nature of the Registrar’s returns as to the *varieties* of fever, although there can be no doubt that the past year showed a high death-rate from fevers. It is difficult to arrive at what is meant by the term. It should only of course include those cases of fever in which the normal diurnal variations of temperature occur without remissions. It is, however, probable that in Ceylon returns

simple continued fever includes all cases of disease attended by fever without any prominent symptom pointing to disease of special organs, and without distinct intermissions.

‘617 cases are attributed to febricula, with only one death. The cause is probably exposure to the sun, and chills when the body is overheated.

‘Ague gives 91,413 cases in the ten years, and remittent fever 3,409, both due to malarial causes. The prevalence of malarial fevers is certainly decreasing. The climate of many parts of the island formerly the most malarious is greatly altered; and places where one could hardly live without the risk of catching deadly jungle fever are now remarkably healthy. Trincomalee shared with some places in India the ill-repute of being the grave of the European, but now there is little ground for this saying. Stations once the most malarious are now remarkably healthy. I may allude here to the disappearance of a disease which at one time was known as the Ceylon disease ‘beri-beri.’ It is now unknown to the present practitioners, and I have never seen a case of the disease in the island. The cause was attributed to the slow action of malaria. The remittent fever of Ceylon is certainly milder than that of India. Typhus, dengue, and relapsing fevers are unknown.’

With the view of ascertaining the nature of the fever that prevails in the Mauritius I sought information from two medical officers of large experience in that colony, and the following is the information I received from them. Even here there seems to be a difference of opinion as to the nature of the fever and its etiology. Dr. Davidson thinks the fever is the true typhoid. Mr. Lovell rather inclines to the belief that it is climatic in its origin.

Dr. Davidson writes :—

‘The etiology of typhoid, whether in tropical or temperate

climates, appears to me to deserve fresh investigation, and its relation to malarial fever certainly demands consideration.

‘The following facts may be of interest to you:—

‘Typhoid fever is believed to have appeared for the first time in Mauritius in the year 1838, and is supposed to have been introduced by the 35th Regiment, in which the disease is said to have prevailed on their arrival in Port Louis. If it could be positively ascertained that the disease from which the 35th suffered was genuine typhoid, and if it were established beyond doubt that typhoid did not exist before in the colony, a very important point in the etiology of the disease would be established. As I have not seen the original records of the cases, I am unable to judge how far the generally-received opinion is supported by evidence.

‘This, however, is past all question, that typhoid fever was exceedingly common in Mauritius from about that period (1838) up to the outbreak of malarial fever in 1866–67. From that time it has given place to the reigning epidemic. So rarely indeed is it now met with, that many medical men hold that it has become extinct. The pathological preparations left by me in Mauritius, obtained from fatal cases occurring in the civil and military hospitals, while I had charge of the former, prove that, although rare at the present day, it is by no means extinct.

‘It has been remarked by several writers on the subject that a relation of *partial exclusion* obtains between typhoid and malarial fevers. Hertz (Ziemssen’s ‘Cyclopædia’) has observed this in Holland, but nowhere has it been so marked as in Mauritius. I believe that such a relation is a fact, and the fact has received two explanations, viz.—(1) that the immediate causes of these diseases, whether they be organised germs or otherwise, are *antagonistic*; and (2) that they are *identical* or closely related. Colin, in his work on “Fièvres Intermittentes,” gives a number of cases illustrating the development of typhoid fever from malarial influences, or rather the passage of what he looked upon as true malarial fever into typhoid. Cases similar to those which he relates have come under my own observation and are doubtless quite familiar to you. I do not see my way to accept Colin’s view of the etiology of these cases.

‘Typhoid fever is exceedingly common in the capital of

Madagascar, situated about 5,000 feet above the level of the sea, in a district free from malarial fevers and dysentery. So common indeed is it that I have the notes, clinically complete, of many hundreds of cases treated by me in hospital during my thirteen years' residence there.'

Mr. Lovell writes :—

'In compliance with your request, I write these lines to express my views respecting the cause of the disease usually termed "typhoid fever" (and which is at all times of the year somewhat prevalent) in Mauritius.

'During my residence at Sierra Leone I frequently observed cases bearing a strong resemblance to true typhoid, but I invariably noticed this distinction, viz., that in the Sierra Leone cases I could never discover any eruption, nor on making a post-mortem examination did I ever succeed in finding any ulceration of the intestines. At that date, 1873 to 1877, I formed an opinion that these cases were due to malarial poison, and from what I have since observed during my residence at Mauritius, I am inclined to believe that the disease usually termed "typhoid fever" in that colony is due to the same cause.

'If you wish, I will procure you carefully-recorded cases of this disease as it occurs at Mauritius, with temperature charts, &c. I should state that the general opinion amongst the medical men of Mauritius is that the disease in question is true "typhoid fever." It occurs amongst all classes of the community, and cases are frequently admitted into the Civil Hospital from vessels lying in the harbour. The bulk of the Mauritian population is composed of Indians (chiefly coolies from the Madras and Bengal presidencies and their offspring), some 2,000 Europeans, about the same number of Chinese, and, roughly speaking, 100,000 coloured creoles (a mixture of European with Indian and African races).'

Dr. Manson, of Amoy, China, has recently sent home¹ an account of an epidemic of continued fever which occurred in China. It was of a circumscribed character and presented anomalous features. In some respects it resembled enteric, in others malarial fever. Quinine in some cases was of benefit, in others it failed.

¹ China Imperial Maritime Customs Medical Reports, 20th issue, 1881.

Dr. Manson remarks:—‘It is very evident that the clue to the proper classification of tropical fever has not been found, and I do not think it will be found until investigators disabuse their minds of the idea that these fevers must be modifications or combinations of two poisons only, the typhoid and the malarial. We are too apt to assume that we can assign correctly the various causes of diseases, and dislike to say, when asked for an answer, “I don’t know,” or to think that there are forces and poisons in nature of whose existence we are ignorant.’

The fever was of a continued type, with high temperature, up to 105° or 106°, diarrhœa, delirium, and some rose-coloured spots. In some of the cases, he says, ‘the symptoms of typhoid were present; in others they were not, beyond the fact that the fever was continued and was uncontrolled by quinine. In one case that did intermit, quinine had no effect.’

Dr. Manson rejects the theory of the combination of typhoid and malarial. ‘The truth is,’ he says, ‘we are nearly entirely ignorant of a number of specific fevers which from time to time affect the inhabitants of foreign countries. I frequently meet with cases of continued fever, both in foreigners and natives, which do not admit of diagnosis and classification—the Tamsuic fever for instance, a disease in which there is continued fever, with pains in the limbs, head, and epigastrium, an exanthematous eruption, no diarrhœa, and convalescence in 20 days or so.’

He asks what were these cases? Certainly not typhoid, certainly not malarial.

‘The physician here has to deal with a miscellaneous collection of fevers whose diagnosis and treatment he has for the most part to make out for himself. A considerable portion of these may perhaps be relegated to what is called “malaria”; but there is a large residuum, examples of which I have given, which can neither be classified among the known exanthems nor among the malarial

fevers. I soon learned to separate them into quinine and non-quinine fevers.' If the fever does not yield to quinine, Dr. Manson considers it non-malarial. Every year he says he meets with such anomalous cases, and has great confusion in his ideas with regard to them. 'One gets little satisfaction from books on the subject. Certain classifications are proposed, but when the attempt is made to attach a name to a given case, the attempt is seldom satisfactory.'

I think there must be many who, if they have looked at tropical fever with unprejudiced eyes—with no theory to support—must concur in Dr. Manson's very candid and truthful comment on this vexed subject.

Dr. Grabham, a Fellow of our College, writes from Madeira, November 16, 1879 :—

'In Madeira, where I have had much experience during the last eighteen years, there are present from time to time cases of typhoid whose origin is precisely that pointed out as referable to general and climatic causes. I have long ago convinced myself that it is vain to seek to trace many well-marked cases of pure typhoid to sources of filth contamination; though these latter abound in Funchal, our chief town, and are frequently associated with their own proper instances of fever.

'The cases which I speak of as having the more vague etiology are those which happen almost every year to some extent, and chiefly in seasons unusually light and sunny, in the persons of young, healthy *new comers*, who roam about to see all our mountain scenery, and succumb to attacks of genuine typhoid fever, quite identical with the typhoid fever of filth, except, as you say, in origin. I have again and again seen such cases arise in situations many hundreds of feet above Funchal, far away from all dwellings, and in regions where the cold drinking water springs from beneath basaltic columns of rock. It is quite certain that, however they acquire it, nevertheless such patients, equally with those who have imbibed their disease from polluted water, are able to spread their infection to others.'

Dr. Ryley writes to me :—

'Three days ago I sent a short paper on this subject, written

last year, before leaving for South Africa, giving the result of sixteen years' experience in this matter in New Zealand, Fiji, Western and South Australia, and New South Wales, after adding that my latest experience of the fever, as it occurred among the troops in Zululand, coincided with my previous experience as above, which is as follows :—

'1. That a fever occurs frequently in all those countries having all the characteristic symptoms of typhoid fever as it occurs in this country.

'2. That in no case, although every pains was taken by me, especially when I held the office of Provincial Surgeon of Westland, have I been able to trace the origin of the fever to contagion.

'3. That in a few cases that disease seemed to have arisen from the fouling of the drinking water by excremental matter in a state of putrefaction. In the majority of cases the fever has broken out among Europeans first inhabiting or cultivating a virgin soil, the "new comers" being most liable to attack, apparently, as in Westland, from decaying vegetable matter, paludal emanations, or other climatic causes.

'I may say, *à propos* of this and in conclusion, that some of the army surgeons and others in South Africa call the fever in question a "hybrid fever" between typhoid and remittent, and treat it as remittent fever with large doses of quinine. When I inquired the meaning of the term, or the reason for its application, I found it was not because the fever presented any remission or differed essentially from typhoid fever as it occurs in this country, but because of the difficulty of accounting under the circumstances for true typhoid by those who hold the views of Dr. Budd or Sir Thomas Watson, or even those of the lamented Dr. Murchison, as to its genesis or etiology.'

Dr. Johnston Ferguson, Surgeon-Major, writes to me :—

'I too have long been of opinion that typhoid fever may have its origin in other causes than the filth to which in most cases it is rightly assigned, and this more especially in tropical and sub-tropical climates. During a service of twenty-seven years I have treated typhoid fever in the Mauritius, Barbadoes, and Bermudas, and this conviction has gained strength as my experience has extended.

‘I have often felt tempted to express my opinion in the medical periodicals, but have been deterred by the difficulty of proving a negative, or of obtaining sufficiently exhaustive evidence to render the position practically unassailable.

‘Being now separated from my notes, and not having by me a copy of my official report on a severe visitation of typhoid fever from which my late regiment (1st Battalion 15th Foot) suffered in Bermuda in 1868-69, I can only give from memory a general outline of the facts, which, however, may possess some interest.

‘The regiment was ordered from New Brunswick to Bermuda in April 1868, as supernumerary to the garrison, to be employed on the works (fortifications). When we left New Brunswick the frost was still on the ground; in four days we were in the commencement of a very hot season in Bermuda. A portion of the regiment was encamped. The head-quarters were in barracks at St. George’s. Dysenteric cases shortly appeared in both parties, followed by typhoid fever in the companies under canvas; and this so severely that the encampment had to be broken up, and the men were put into barracks at Bray Island, and not employed on the works. The fever then ceased. At a later period, another party was encamped at another part of the islands, relieving a party of the 61st Regiment. The 61st party had been free from fever. They were acclimatised. Fever (typhoid) broke out again so severely in my regiment that the party had to be withdrawn. About the same time it broke out at head-quarters, and was very severe. I stated in my report my inability to trace it to any defective sanitary arrangement.

‘In the encampment the dry-earth system of conservancy was adopted. The water used was rain, collected in tanks removed from every possible source of pollution. At head-quarters the tanks were of iron. Every attention was paid to the sanitary condition of camps and quarters. The 61st acclimatised party had been free from fever with the same arrangements in the same encampment. At head-quarters the barracks were favourably situated, above and outside the town, and were exposed to wind from every quarter. The town, with its narrow lanes, dense population, cut off from the wind on two sides by the hill on which the barracks stood, and with certainly inferior sanitary arrangements, was at this time free from typhoid fever.

‘The only universally applicable explanation of these cases

appeared to me to be "climatic causes affecting new comers." A very sad case in support of this view occurred on the relief of my regiment in 1870. A young officer of the relieving regiment, lately married, took a detached cottage, near my own house. It had been occupied for more than two years to my knowledge by an officer of engineers, his wife and child, without any case of fever. The new comers had not been in it two months before this officer lost his young wife of typhoid fever.'

Surgeon-General Irvine, of the Army Medical Department, informs me that a recent outbreak of enteric fever has taken place among our troops in South Africa. I have not received any particulars of cases or autopsies; but the Principal Medical Officer in transmitting the report attributes it to the fouling of the streams on which the camps were pitched by dead cattle and filth of all sorts, the heavy rains washing the foul matter into the rivers. In this case the origin of the disease seems attributable to animal emanations, but not especially to a specific contagion developed in human excrement; such epidemic outbreaks are probably generally to be explained by similar causes. The stations whence the reports of the outbreak have come are St. John's River, Pietermaritzburg, Ladysmith, Ingame, and Newcastle. The strength of the troops was—Officers, 107; men, 2,939; women, 43; children, 69. Of these one officer was attacked, but recovered; 183 men were attacked and 33 died. The women and children escaped. No details having reached me, I am unable to give any account of the symptoms of the disease or of the post-mortem appearances, nor why the women and children should have escaped.

It is said by some writers that enteric fever is a common disease among the natives of India; by others, that it is almost unknown. I think there can be little doubt that it is common enough. The causes which influence the European equally affect the native, and unless it be a race peculiarity, there can be no reason why the native should suffer less than the European.

Dr. Wise's reports from Dacca, Dr. McConnell's from Calcutta, and many others affirm its existence.

The Sanitary Report for Assam, 1880, contains an account of an outbreak among the sepoy's of a native regiment stationed on the north-eastern frontier, which clearly proves the existence of the disease, and in this case it would seem to be traced to defective water. The civil surgeon of Góalpára gives a description of a case in point, and of post-mortem examination, which leaves no doubt as to the nature of the fever.

The Sanitary Commissioner says:—

'The Civil Surgeon of Góalpára records an unequivocal case of typhoid fever, and he states that the diagnosis was confirmed by the post-mortem examination. Early in May three cases of enteric fever occurred in the 44th Regiment Native Infantry cantoned at Shillong. Two were in Goorkha recruits, whose ages were 21 and 22 respectively. They arrived at Shillong on April 26: one was admitted to hospital on May 3, and recovered; one was admitted on May 12, sickened of enteric fever, and died. The other was that of a Goorkha sepoy, age 28, who had returned from the Nága Hills; he arrived at Shillong on May 2, he ailed on May 8, on May 11 he was admitted into hospital, and on June 12 he died.

'As much interest attaches to these cases of enteric fever, it may not be out of place if I subjoin the main features of a case:—

'On May 15 fever continuous, morning temperature 101°·2, evening 103·6; lies in a semi-comatose state, appears deaf, face flushed, tongue coated with a creamy fur, marked tremor of muscles and subsultus of tendons, constant muttering delirium, no sound sleep, abdomen soft and doughy, diarrhoea of drab-coloured homogeneous-looking stools, four or five in the twenty-four hours. Loud gurgling with marked tenderness on pressure over the lower abdomen and right inguinal region. The patient sunk on June 12. The post-mortem examination exhibited the true enteric lesion. . . . In the last four inches of the small intestines two perforating ulcers were discovered, viz., one close to the ileo-colic valve, and one about four inches from it. The nine uppermost Peyer's patches apparently healthy, the next patch in a state of rose-coloured congestion, the next two plainly

infarcted, and the remaining, seventeen in number, as well as the numerous solitary glands, were in a state of ulceration more or less ragged. Two patches near the valve were perforated. Two circular ulcers, the size of a four-anna piece, were found in the upper part of the colon.

'Seven cases were recorded, and during the occurrence of the disease every precaution was adopted; excreta were removed, and the drinking water as far as possible protected. The men were all Goorkhas, and meat-eaters. One man was 35 years of age; the total average age of the other six was 23·3 years. The first three cases occurred in men who had recently come to Shillong; Nos. 1 and 3 arrived on April 26, from the Nepál Frontier, viâ Gorruckpore, the regimental recruiting ground of the N.W. Provinces. No. 2 arrived on May 2, six days later, from the Nága country. Previous to their arrival, so far as it was possible to ascertain, there had been no manifestation whatever of the disease, either among the troops in any part of the cantonment or among the civil community. The lines the regiment occupied are on a hill, open and airy; they are quite apart from the civil station, and they are separated from the other part of the cantonment, then occupied by the head-quarters of the 42nd Regiment, by a deep mountain gorge. The water supply of the lines is from a hill-stream brought in at some distance by an open channel; but as it passes through other premises, and supplies two or three private families before reaching the lines, it is undoubtedly open to contamination. The sanitary state of the lines was very fairly good, clean, and by natural slopes well drained.'

' Outbreak of Enteric Fever among the Civil Community.

'Besides the cases above recorded, thirteen other cases came under observation of the same medical officer of the 44th Regiment, between the months of July and November, among the civil population. They are noted in the subjoined table:—

Number of Cases	Class of Resident	Remarks
1	English	Residing in Cantonment
1	Eurasian	" " Laban
1	Hindu prisoner . .	" " Gaol
6	Khásias (hill tribe) .	" " Maokar
4	" "	" " Villages

‘The Khásias are hill people; and all, except the Hindu prisoner, were flesh-eaters. No very accurate account was kept of these cases. But the notes which were taken show clearly the morning and evening temperature, hæmorrhage from the bowels, tenderness over the lower abdomen, muscular tremor as symptomatic and characteristic of typhoid fever. Five cases were fatal, but no post-mortem was permitted except in the case of the Hindu prisoner, in whom the enteric lesions were fully developed. Three of these people lived under good sanitary conditions; but the sanitary conditions of the Khásias were less perfect and their water supply less pure. The water supply of the 44th Regiment had no connection whatever with that of other localities in which typhoid fever occurred. Out of the twenty people who suffered, seven dwelt in the regimental lines of the 44th Regiment, one in the civil station, one in the gaol one in the suburb of Laban, six in the Khásia village of Maokar, three in the village of Dombynka, one in Manli, localities widely separated and supplied by water from different sources, having no connection with each other whatever. Nor did any of these people in the civil community live in houses contiguous to each other.

‘No record of any such previous outbreak nor of any case for years past exists. The Civil Surgeon remarks: “Years have passed in which no case has come under my observation.” But even if we suppose that cases may have occurred and passed unnoticed, which is not improbable, it is quite clear that there has been no such previous outbreak as that described, and which occurred in this station and its neighbourhood between May and November last.’

There can be no doubt I think, whatever view may be taken of the etiology of these cases, that they clearly show that under certain circumstances natives and Europeans are both affected by fever with enteric symptoms. It is by careful study of such outbreaks and of isolated cases, as regards the locality, water, sanitary conditions, previous history of the sufferers, and their personal attributes, age, race, temperament, time of year, &c., that we may hope to discover the true causation.

But while the returns of the Sanitary Commissioner, the statistics of the hospitals, and other records, place the

existence of this form of fever among natives beyond question, the etiology is still uncertain. My own impression is that it may depend on specific organic poisoning, but I doubt if this can be limited to human excreta; and believe that miasmatic poisoning under certain undefined modifying circumstances may give rise to continued or continuo-remittent fever, which becomes practically indistinguishable from specific enteric fever.

That enteric fever should have one specific origin in temperate climates is no proof that it may not have others in the tropics. Some of the low forms of fever described by older writers in this country, and by modern ones in France and America, as typho-malarial and paludal-typhoid, seem to be of this nature; and in this connection Dr. J. Harley's views on this subject, as referred to by Dr. Wise, seem to me of great interest.

It is worthy of note that the apparently increased proclivity of the meat-eating and spirit-drinking races to enteric fever is calculated to awaken attention to the possible effect of the diet of the European soldier in India; but though it may be more frequent among, it is not confined to meat-eating natives, as shown by Dr. Wise's cases. As regards the etiology and causation of enteric fever much uncertainty unquestionably exists. Even in Europe, where its connection with a specific poison, or with the products of faecal or other organic decomposition, contaminating air or water, seems to be established by a large number of observations, the cumulative effect of which amounts to very forcible evidence, it cannot be said that we have exhausted our knowledge of causation.

I think I have cited evidence enough to show that in the opinion of many medical officers of experience ulceration of the small intestines and Peyer's glands is not necessarily indicative of specific enteric fever, but may also occur in the climatic fevers of remittent and continued type. I believe a considerable amount of climatic fever occurs in the tropics, in which the symptoms and phenomena so closely resemble those of true enteric fever

that they may cause one to be mistaken for the other, and that the post-mortem discovery of ulceration attests the severity of the disease; but this does not proclaim its original cause. I would ask medical officers in India to study each case in all its aspects most critically, for no one will deny that we have still much to learn about fever in tropical climates.

All I have said indicates the existence of fever which is neither simple continued, ordinary remittent, nor specific enteric, and that it should stand apart, its affinity being with malarial rather than with true enteric fever, though it has much in common with both. It may, after all, turn out to be true enteric fever, and were it admitted that the specific form of fever may originate in animal effluvia and emanations generally, and is not restricted to a specific contagium developed *only in human excrement*, then it is probable enough that in such a source we may find its origin, for there are few localities in which organic emanations do not taint the air or pollute the soil or water. Still the fact that these conditions are often rife, and yet this form of fever so infrequent, whilst the effects of season, locality, and age, play so important a part in developing it, seems to show that climatic conditions are largely concerned.

Dr. Woodward's typho-malarial seems to me most nearly to describe the form of fever, but I confess to some disinclination to adopt a name which indicates hybridity, and would rather accept Professor Léon Colin's view of transformation, though even this mode of describing it is hardly appropriate; for whilst the general aspect of the disease is that of specific enteric, careful examination of the previous history and onset may enable the observer to assign to the fever its true designation.

There is no reason to doubt that certain causes produce the same effects in India as elsewhere. I have seen and treated many cases in India which I never thought of assigning to other than a specific cause, whether that was to be looked for in a specific contagion derived from

another individual's bowels, spontaneous evolution, or the decomposition of organic matter. But I believe that typhoid symptoms, diarrhoea, and enteric lesions also occur in other forms of tropical fevers, probably due to organic miasmata combined with those influences of earth, air, or place that are developed most readily in tropical or sub-tropical climates, but which, under certain conditions, as pointed out by Pringle, Woodward, Colin, Gordon, and others, produce similar effects in other climates.

To summarise the facts about this form of fever as it occurs in India, let me turn to the latest reports and see how the matter is viewed at present. The preponderance of opinion is in favour of a specific origin for all enteric fevers; but there is a certain vagueness as to the etiology, varying from the specific contagium to the results of decomposition of organic matter generally, including malaria. Some consider that it is due neither to specific contagion nor miasm, but to certain climatic conditions. No one, I think, disputes the *existence* of enteric fever; and I doubt if any exception would have been made to the name '*enteric*' did it not imply a specific contagion, conveying views of specific causation which are not certainly of general application. It suggests questions of importance in regard to the right age, time, and season for sending soldiers to India, to say nothing of the hygienic questions arising out of causal relations. The Sanitary Commissioner's Report of 1877 says that out of 233 cases of typhoid, 92, or 39 per cent., proved fatal; the admission rate being 4·1 per 1,000 of strength; that 2·45 per cent. occurred at or under twenty-four years of age; 1·55 at twenty-five to twenty-nine; 0·99 at thirty to thirty-four; and a few or none above that age—showing that the disease tells most severely on the younger men. Bryden, in his *Statistical History of the European Army in India up to 1876* (published 1878), says: 'It (enteric fever) has no geography; and it is a matter of popular observation that no regiment or battery escapes enteric fever *in the first year*, whatever cantonment of India may

be selected.' 'Out of seventy-three bodies of men, two regiments and seven batteries only returned no case of enteric fever in the first year.' And he gives the following analysis of 368 deaths that occurred between 1823 and 1876 :—

Ages	Total deaths
24 and under	255
25 to 29	90
30 to 34	17
35 to 39	4
40 and upwards	2

Seventy-five of these deaths occurred within three years after landing in India, and 94 per cent. of the total were among men under thirty years of age. Bryden further says that out of 132 deaths from enteric fever in 1878, 90 occurred in men who had been under twenty-two months in India. This shows that youth and the first year of service in India are the great predisposing causes.

A most valuable report has recently been drawn up by Brigade-Surgeon J. Marston, the able and accomplished secretary to the head of the Medical Department in Bengal. He has had ample opportunity of seeing the disease and of becoming officially acquainted with all its manifestations, after long experience and careful study of the subject at home. The following is the purport of his remarks to me after some years of Indian experience :—

'I came out here imbued, rather than otherwise, with a belief in the truth of the views of European pathologists, but Indian experience has compelled me to recognise that those views as to the causes of enteric fever are too exclusive, and quite inadequate to account for the facts; they do not cover anything like all the facts, and they are irreconcilable with some of them.

'Of course if a man desires to view everything through the spectacles of prejudice or preconceived notions, he will discover probably out here what he seeks to find. But how can any specific or other fæcal contamination of air, milk, or water account for such facts as these?

'I. The remarkable proclivity to this disease exhibited by recent arrivals in this country.

‘II. The occurrence of cases at certain seasons at stations extending over vast areas of country.

‘III. The isolated nature of such cases in a great many instances.

‘IV. Their occurrence at certain definite seasons, *e.g.* at the hottest and driest, when the wells are lowest, and at the end of the monsoon, when they are highest. And you know what a rainy season is, how it would in a short time carry everything (let alone faecal germs) from the Himalayas to Calcutta or Kurrachee, according to which of the big river courses it took.

‘V. Again, from the British dominions in India up to Kabul you had at almost all the military posts occupied by the various columns in Afghanistan cases of enteric fever, notwithstanding that many of these posts must have been occupied by Europeans for the first time in history.

‘I have tried unsuccessfully, and others have done the same, to obtain reasonable proof out here of the operation of those causes *set down as the only and invariable causes of enteric fever at home*. Of course I do not say such causes do not exist and may not be followed by the same effects out here. It is very likely that faecal contamination of air or water, or infection, may account for outbreaks in India, but a man would indeed have a hard task to account for all the cases in India on any hypothesis of the sort. The more I think of it the more convinced I feel of the inadequacy of the usually accepted views to account for all, or anything like all, the facts in this country; nay, I go further, and am very sceptical as to their accounting for all the facts elsewhere, in France, America, and Britain.

‘I wish I could say positively what does cover all the facts, but I cannot. I have, however, hazarded a hypothesis.’

He says also:—‘While fully recognising, then, the force of the evidence on which European pathologists dwell in regard to the specific nature of enteric fever and its connection either with specific or filth-causes, it is impossible, on the other hand, to resist the evidence that such causes fail to embrace all the facts observed in this country. It may be urged that when confronted with the positive observations of repeated occurrences in Europe negative evidence is of very little value, amounting, as it does, to an inability to trace the continuity of a chain of causation,

the connecting links of which are necessarily invisible : but the well-nigh universality of the disease here (India), its connection with the age and recent arrival of its subjects, and the seasonal regularity of its appearance, its continuance and disappearance in a newly-arrived corps within definite limits of time, are in themselves positive evidence—facts that must be reconciled with any theory which claims to be true for India as well as Europe. They point to a remarkable susceptibility to the disease at any rate on the part of young men during their first year or two of Indian service, and this compels us to inquire whether there may not be something in the climate itself and the new conditions of life here (India), irrespective of any specific cause, or any filth cause even, capable of originating the disease in question.

‘ On the whole, I think, as a tentative and working hypothesis, that there are two forms of it in India, which cannot, however, be clinically or pathologically differentiated : one (the larger class) which does not depend on the contagion of any specific poison generated in the intestine of one person and conveyed to another through some vehicle, nor indeed on any fæcal poisoning, or poisoning of any kind, unless it be that the patient is autogenetically poisoned by his own fæcal matters ; the other, occurring in outbreaks (not singly, in isolated cases) and with a history by which the cases can be traced to some common cause, such as infection, fouled air or water, diseased or tainted meat, &c. These attacks occur in communities at one and the same time, or following one another quickly among men of the same corps and placed under the same conditions ; and the ages and length of tropical service of the men are often very different to those of the other variety. Occurring under the circumstances stated, age and service are indifferent elements ; within the limits of a soldier’s age any man may be attacked, though the young and recently-arrived are more predisposed.

‘ In the first variety, however, I am disposed to think climate—meaning by it to include the whole combination

of changed physiological conditions environing the young and newly-arrived soldier in this country—plays a very important, if not the main part. It is a notable fact that, of a number of fatal cases returned as remittent fever, where the post-mortem appearances disclosed an absence of any intestinal glandular lesion, the subjects are older men and longer resident in the country; whereas where ulceration of Peyer's patches was found, and the fever has been diagnosed as remittent or enteric, the subjects are, as a rule, younger and less long resident soldiers.

‘Of course it may be that there are specific disease-germs present which operate under certain meteorological conditions and at certain seasons only—in air, soil, or water—and that susceptibility to their action is vastly increased in new arrivals, a relative or absolute immunity being brought about by longer-continued exposure to them. This is an hypothesis required if we are to accept the views of each disease depending upon the action of its own, or its own variety of germ.

‘Of the following facts there can be no doubt:—

‘I. Given a population within the limits of the enteric fever age, recent arrival in India is a powerful factor in its production.

‘II. The development of enteric fever at certain definite seasons, corresponding with heat seasons, in isolated cases, or groups of cases, according to the existence of the material, at numerous stations extending over a vast distance.

‘III. That these seasons are not the malarial seasons, nor the places malarial places especially.

‘IV. That the disease so occurring is clinically and in its morbid anatomy allied to, if not identical with, enteric fever observed elsewhere.

‘V. That outbreaks and local epidemics of fever marked by the post-mortem lesions of enteric fever are practically unknown, or have not been recognised among the native population.

‘The deaths from enteric fever among young European

soldiers during the first two years of service and at different ages were:—

Cause of Death	Deaths per 1,000 of strength in biennial period		Rates of Liability in percentages	
	Under 24	25 to 29	Under 24	25 to 29
Enteric fever	9.7	10.16	44.31	46.08

‘It would follow from this that between twenty and twenty-five the chances of dying of enteric fever are not very different.

‘The question of causation is important; if the enteric fever be a specific fever depending on a specific poison, or if it be essentially a filth-fever originating spontaneously from pythogenic causes, then we should be able to limit the spread of infection, or by sanitary operations exclude it from military cantonments; thus every precaution is taken, but it is impossible to exclude all possible sources of origin of the poison. The men are not always confined to barracks, and have access to native villages, houses, &c.’

Marston seems to doubt the existence of enteric fever among the native population, but, as I have shown, it does exist abundantly, and were post-mortem examinations more readily obtainable, it is probable, I think, that the characteristic lesions would be found, and not unfrequently.

The medical authorities have given instructions to investigate all cases most closely. But ‘medical officers have utterly failed in India to satisfactorily trace out the intimate connection of *the disease with filth-causes of specific infection*, with which, according to European authorities, it is invariably connected. Such failure has not been due to any want of zeal on the part of the medical officers, who have striven to harmonise the conviction due to the doctrines in which they have been educated with the results of their Indian experience.’

The soldier on landing in India is placed under entirely new physiological conditions in regard to climate, food,

and mode of life. The separate influence of some of these it would be difficult if not impossible to determine, but within the term climate are embraced heat and soil, and under the latter (soil) we may include malaria. It seems quite clear from the tenor of this report that the conviction forces itself inevitably on the medical authorities, that the causal relations of this form of continued fever are not, in India, limited to those which give rise to it elsewhere. It is in this doubt that I share, and I feel convinced that there is at all events sufficient ground to give interest to searching inquiry, which will probably show, not that there are more fevers, but more causes than have been believed. But I would, for the sake of precision of registration, urge the adoption into the nosological returns of some term that will distinguish climatic from specific fever.

It seems hardly necessary that I should detail the symptoms of enteric fever, but, as in the case of agues and remittents, I shall sketch them briefly. I shall not attempt to draw any clinical distinction between the continued fever with enteric symptoms and the specific enteric, for I am not aware of anything that could be regarded as absolutely pathognomic, though closer observation might enable the observer to differentiate them. The history, the circumstances, and the personal peculiarities of the individual would probably give a clue to the etiology. I am aware of the stress laid on the quinine test. Quinine is likely to be useful, both for its anti-periodic and its apyrexial properties. Supposing the fever to be of the specific kind, quinine will be of benefit as an antipyretic. I have, too, often known it fail to do good in an undoubted malarial paroxysmal fever on the one hand, and on the other seen it so effective in reducing temperature in ordinary pyrexia, that I cannot accept it as the infallible test that shall clear up the doubtful etiology of an obscure fever.

Marston, struck no doubt with the varied phenomena presented by fevers in India, says: 'There can be no

doubt that a patient admitted with the symptoms and history of ardent fever, and exhibiting a higher range of temperature in the first and second days than is usually witnessed in enteric fever, as well as a patient with the symptoms and rapidly acquired maximum temperature of ague, may go on to exhibit the symptoms, and run through the whole course of an enteric fever of pronounced type.'

The symptoms are much the same in India as elsewhere, modified perhaps by malarial influence, which as it so frequently colours and modifies other disease, it is not strange that it should do so in this. It has been thought that any difference the disease may present in India, as compared with it in England, is due to the added action of malaria. There are characters which differentiate enteric from remittent fever in the early stages, though it is often exceedingly difficult if not impossible in the more advanced stages to distinguish them.

At the outset it may be as insidious as in England, and for the first few days there may be only malaise, chills, perhaps diarrhoea, loss of appetite, weariness, aching of the limbs, and headache. The patient at length lays up, the pulse quickens, the skin becomes hot and dry, there is thirst, heaviness, and dullness, whilst the thermometer indicates a temperature gradually rising until it reaches 104° or more, with a remission towards the morning. The abdomen becomes distended, and there is tenderness on pressure, especially in the right iliac region, with gurgling. The diarrhoea probably increases, and becomes of a yellowish colour; it may be tinged with blood. The tongue is red at the tip and edges, dry, cracked, and tender. The teeth begin to be covered with sordes. During the second week the characteristic spots make their appearance, though they are often absent, and on the dark skin of the coloured races are difficult to detect.

As the disease progresses the patient becomes delirious; the delirium is at first a wandering, but it gradually becomes incoherence, and it may be noisy or

muttering, with complete prostration. The diarrhœa increases, the tongue is dry and glazed, the teeth covered with sordes. There may be epistaxis, or hæmorrhage from the bowels, and the patient becomes quite unconscious. The temperature rises to 106° or even higher. He has subsultus of the tendons, muscular twitchings, picking of bedclothes. Death supervenes from exhaustion—the result of the disease, or from peritonitis caused by perforation of the ulcerated bowel.

The ordinary duration of the fever is three weeks, and it is often longer; in severe cases it may terminate fatally much earlier, probably before the intestinal ulceration has taken place, by the intense action of the poison on the nerve centres; but in milder cases it may also terminate earlier, and it not unfrequently happens that about the fourteenth day the symptoms improve, the temperature begins to fall, the general symptoms abate, the diarrhœa decreases, and the appetite and sleep are better.

The diagnosis between specific enteric and climatic enteric is often very difficult. The close resemblance between some remittents and the specific form is very great. It is by observation of the earlier symptoms and study of the previous history that the distinction will be practicable. In the specific form the invasion is gradual, and it is not for some days, during which the temperature rises in the evening, until about the fourth evening, that it attains to 104° . In the climatic or malarial forms the premonitory symptoms are more sudden. There is more marked chill or rigor, the malaise is greater, the temperature rising to 104° or 106° as early as the evening of the first or second day. These distinctions are not always well marked, and it may be quite impossible to establish these points of diagnosis. There is diarrhœa in both, and all the other symptoms, ulceration being established, become identical. The rose-coloured spots are by some regarded as pathognomic, but they are often observed in cases of specific enteric, and it is very difficult to detect them on the dark skins of natives; it is quite possible

that they may, standing in relation to the bowel ulceration, occur, however that condition is established. The premonitory stage is of a different character, the onset being more sudden, and ushered in by chills or rigors, the temperature rising above 104° often on the first day.

There is, as in all fevers of malarial origin, a disagreeable sensation of chill from contact of air, and even cutis anserina when the body temperature is very high. Dr. Wise has observed that the stools are always acid; to this he has paid much attention, and he thinks it a point of considerable importance. He further remarks that the eyes are always bright and glistening, and the anxiety depicted on the countenance of the specific typhoid patient is wanting. In regard to the state of the tongue, eyes, and urine he had not formed any definite conclusion.

Dr. B. Browne, of Lahore, has noted the points of distinction between remittent and typhoid in a paper in the 'Indian Medical Gazette,' September 1879, and adds that an important means of diagnosing these diseases is the treatment by quinine. In remittent fever, if large doses of quinine be given, the fever will in most cases be cut short, which is not the case in typhoid. Quite true, but there is not much danger of confounding a remittent with intervals of well-pronounced remissions with typhoid. The cases in which there is difficulty are the continued or continuous remittents, and there quinine will not cut short the fever, though it will reduce the temperature, and for this reason is a most valuable remedy. In fever with enteric ulceration, however caused, it is not to be expected that it could be cut short, and therefore quinine cannot be regarded as the crucial test, though in the earlier stages, before ulceration has set in, it certainly may prove so. It is on points of detail of this kind that further observation is required, and I would ask our colleagues in India and the tropics to consider this among other desiderata; for it is in the study and careful observation and com-

parison of these special features that the main issues will be determined. General descriptions abound, and it seems that all that is wanted is further investigation, with the absence of all bias in favour of this or that theory, of the facts bearing on the etiological and pathological relations of these fevers.

I have already referred to the great fatality of enteric fever among our young soldiers during the early part of their service. It is indeed the great fever death-cause among British troops in India. A certain amount occurs in the civil population, and generally, though not always, among young people. Every year I had such cases of marked enteric fever, with all the characteristic phenomena. In none was there any special ground for attributing it to *fæcal* sources, but of course it is impossible to say that they did not exist. The worst, and a fatal case, that recurs to me was one of a gentleman nearer fifty than forty years of age, and in whose condition and mode of life it would be difficult to trace a specific origin of the disease. I regarded the cases generally as ordinary examples of enteric fever as it occurs in England; but I never could feel satisfied that the origin was quite the same, unless indeed organic miasmata be allowed a wider extension than that depending on *fæcal* matter: were this admitted, causation would not be so far to seek.

It is in the earlier stages and onset of the fever that its true etiology may be detected—commencing insidiously, slowly, and with a gradually rising temperature and slow development of the abdominal symptoms, and nervous prostration. Especially when it is observed in large communities, in cities and camps, the likelihood of a specific origin seems probable, though I would ask to extend the range of causation beyond mere sewage and *fæcal* contamination.

But when it occurs in the course of simple ardent or paroxysmal fevers, when the rise of temperature at the outset is more abrupt and sudden, and when the thermo-

graph is irregular, I suspect that the origin is to be sought in something more general than a specific faecal source. I admit the extreme difficulty of differential diagnosis after a certain stage, and when intestinal ulceration has taken place, and can well imagine that septic absorption from these ulcerations may so modify the symptoms that there is practically no real distinction.

In short, I believe, as I have before said, that in India enteric lesions are apt to come on in the course of miasmatic fever, and that in this condition they not only resemble but become identical with those of specific enteric fever, which is caused in India as in England. If asked, Why seek for any other explanation than that accepted in this country? I reply that in India the facts are not covered by the explanation; and that there is more evidence that ordinary climatic fever may assume the typhoid, *i.e.* enteric condition, than that all enteric fever is caused by faecal contamination.

I venture to think that this view will be taken by others who have the opportunity of extending their study of fevers in India and the tropics.

It is not necessary that I should enter more into the symptomatology of the various phases assumed by Indian enteric fever. As I have mentioned their chief characteristics, it would unduly prolong the subject to do so. Nor is it necessary to say much on the subject of treatment, for in fact it is exactly that which is adopted here, and consists mainly in the careful administration of fluid nutrients, and avoiding all that could excite or irritate the disordered bowel. Diarrhoea should be controlled, not unduly checked; and temperature should be reduced by apyretic or diaphoretic remedies or by baths. Quinine in moderate doses, whilst especially indicated in the fevers of a miasmatic origin, is highly useful in pyrexia, however caused, and the anxiety so often expressed about the expediency of giving it is needless; whilst as to the mode and extent of its administration, the

circumstances of each particular case will be the proper guide. As regards wine or alcohol, I have generally found it of great use, and have administered it according to the effects it produced, seldom having had difficulty in ascertaining what these effects were. As to nourishment, animal broths and milk—perhaps diluted with some alkaline water—are the most appropriate; and I have always been impressed with the necessity of avoiding any possible source of gastro-intestinal irritation, even after convalescence was well established. Relapses occasionally occur, and a nearly fatal one in the case of an officer who, in the fourth or fifth week suffered from a recurrence of dangerous symptoms, the result of eating a few raisins given him by the nurse, left a strong impression on my mind of the importance of caution as to diet. The temperature charts show the varied character of the pyrexia, and how little reason there is to draw a distinct line of demarcation between the different forms of fever. I regret that I am unable to analyse these charts at length; I will only ask you to look at them and the specimens on the table of pathological lesions from fever patients in India, for which I am indebted to Professor Aitken, of Netley—to whom, as to many of my brother officers in India and at home, I am under great obligations, as my frequent allusions to their contributions and the cases they have sent me, attest.

I am reluctantly compelled to bring these lectures to a conclusion. I knew the subject was extensive, but it was only on attempting to compress it into the short space allowed that I realised the magnitude of the work I had undertaken. I am sensible I have omitted much that should have been said, and have but imperfectly availed myself of the time at my disposal. I had hoped to have considered the subjects of typhus, relapsing fever, dengue, and Indian plague; but these for the present must be

deferred. It only remains for me to thank you for the attention with which you have listened to my imperfect endeavours to add something to the story of Indian fevers.

CASE XLVI.

REMITTENT, WITH LIVER ABSCESS.

(Dr. Hoystead.)

‘ A recent case reminded me of the conflicting views held on the close affinity which exists out here between remittent fever and typhoid.

‘ A young soldier was admitted into Base Hospital, Kurrachee, for colic, due to chill, a fruitful source of disease during the monsoon months. In four days’ time his disease was changed to remittent fever; evening temperature 104° and a morning one of 101° , demonstrating morning remissions but a continuous fever. I subsequently took over the case and changed the name of his disease to typhoid fever, owing to subjective symptoms and the lowering of the temperature, which became fixed at 102° all through the twenty-four hours. He complained of localised pains in the iliac regions, and generally lay with his legs flexed; his tongue became dry and brown, complete with anorexia, and an eczematous rash appeared upon his face, nose, eyelids, and scalp, which commenced as bullæ and vesiculæ. Bowels generally normal, occasionally very loose. He died from failing power and syncope, due to a large loss of blood per anum.

‘ *Post-mortem Examination.*—Right lung congested from upward pressure of the enlarged liver; pulmonary organs otherwise healthy. Liver much enlarged, and contained one large abscess. Spleen a little enlarged but remarkably healthy in appearance. Kidneys slightly congested but normal in size. Intestines from the cæcum down to the upper portion of rectum studded with ulcers of all shapes and sizes, and one of which had perforated the mass of upper portion of rectum without being attended by extravasation or peritonitis.

‘ The question remains to be asked what this man died of—malaria or enteric fever? I am strongly disposed to think that malaria is frequently responsible for the destructive changes in the bowels which physicians attribute to specific typhoid in a climate.

'The etiology of the disease was closely investigated, with negative results for the specific disease. I could not detect the characteristic rose spots of typhoid.'

[The liver abscess and dysenteric ulceration remove this case from among the category of fevers, but it is interesting as showing that their affinity is recognised by Dr. Hoystead.—J. F.]

I am indebted to Dr. Maclean, C.B., for the following interesting case :—

CASE XLVII.

MALARIAL FEVER AND LIVER ABSCESS.

'Mr. M. has been in every bad climate in the world in the course of his business, and is super-saturated with malaria. He has overworked and been subjected to great mental strain for a long time. After numerous attacks of fever he was put on board ship at Rio in a very exhausted state. On the voyage he had a severe attack of dysentery; when I took charge of him he was in a state of extreme prostration, but the dysentery, which had been judiciously treated by a retired army surgeon on board, had ceased.

'He was emaciated and very anæmic; had a daily paroxysm of fever, without rigors, but followed by exhausting sweats. The morning temperature was seldom under 100°, rising after 4 P.M. to 101° or 102°, and occasionally a degree higher. His appetite was capricious and his bowels irregular, the motions being at times firm, at others pultaceous, and always with a marked absence of bile. No dysentery, no diarrhoea. Urine scanty, 'febrile,' no albumen, no sugar, giving no bile reaction, but loaded with lithates. There was no abdominal tenderness; a moderate amount of tympanites, and no trace of tenderness in the hepatic region. But the hepatic note was distinctly deficient in the mammary line, hardly so in the axillary, and still less in the dorsal lines. I satisfied myself by repeated examinations in different positions of some liver contraction, in, as I have said, the mammary line, which I was able to connect with spirit-drinking in early life, a habit long abandoned, at least by the patient's account.

'He was placed on full doses of quinine for some days; little effect was produced on the daily febrile attack until the chloride of ammonium in xv. gr. doses three times daily and a two-grain pill of euonymin every night were taken. The effect of these

on the liver was striking. They roused it into action, and when the above had been used for some time the temperature began to decline, and as the quinine disagreed it was discontinued. For about a week three grains of potass iodid. was added to the chloride of ammonium with good effect.

'After a time he was seized with violent neuralgic pain of the supra-orbital nerve, which only yielded to remedies local and general long persevered in. Arsenic, so useful in such affections, I could not use for fear of lighting up mischief in the still tender mucous membrane of the bowels. When this yielded there was a transfer of the pain to the abdomen, as distinctly paroxysmal and neuralgic as the other, and aggravated by a certain amount of tympanites. Nothing for a long time relieved this, but first morphia by hypodermic injections, and when this failed, laudanum by the mouth. At last I tried turpentine in ten-drop doses, the effect of which was very notable. I have seen him, when this pain was at its worst, rolling on the floor and declaring he would shoot himself. I tried iron in full doses, but it did harm. I was tempted to this by his anæmic condition, and I still think that by-and-by some preparation of it in the dialysed form will be called for.

'He is now in all respects better, still very weak, with slight returns now and then of the abdominal neuralgia, and the temperature is daily approaching the normal, morning and evening.'

I saw Mr. M. on March 2, 1882, in London. He was weak and seemed exhausted; he had recently come from Southampton; said he had suffered from agonising pain in the abdomen on different occasions but was easier then. He looked pallid, worn, anæmic, and saturated with malaria. His bowels were confined. I gave the nurse instructions, and told her whom to consult during my absence, as I was going out of town for two or three days.

He had been taking frequent doses of laudanum. I told the nurse to withhold it except under medical sanction.

A telegram reached me at Bournemouth, saying he was in great suffering.

On Monday I saw him about 2 p.m. He was relieved from pain, and was talking to friends; but he looked feeble, his breathing was shallow and hurried, and his pulse feeble. There was no pain on abdominal pressure, but he was very impatient of any examination. I saw him at 6.30 p.m.; intense agony had come on again; he was very ill; pulse quick and feeble; skin clammy, breathing hurried, shallow, and distressed;

stimulants, warmth, opium, and chloroform were resorted to, and he was again relieved. I remained with him till 9.30, having the advantage of Dr. L. Branton's advice, when Dr. Selfe Bennett took charge of him; his account of the case is appended. The intense paroxysmal pain, the condition of pulse, respiration, and temperature, which became subnormal, and his previous history, suggested malaria as the cause of his suffering. An abscess of the liver had burst and was slowly draining into the cavity of the peritoneum. The coagula contained in the cavities of the heart and pulmonary artery terminated his life perhaps sooner than might otherwise have been the case, and caused the apnoea from which he suffered.

'At 9 P.M. on March 6, 1882, he was recumbent, with his head and shoulders raised; his respiration was extremely rapid; his pulse easily felt, but uncountable from its running character; his face was sallow; his lips bloodless; his aspect showed great distress and anxiety, and I thought him dying. His complaint was of intense pain over the site of the descending colon. About 10 o'clock the bowels acted, and he passed a small and moderately loose motion, of a dark colour. After this he sat up for a few minutes, and walked (with support) from the sofa in the front room to the bed in the back room, where he lay with legs straight, and refused to be covered with more than a sheet and one blanket, feeling hot, though his skin was cool and moist to touch; he said he felt better, his respiration was slower, he could speak with a clear and distinct voice, his pulse was 90, small and feeble. He turned on his left side after drinking some milk, soda, and brandy mixed; his tongue was coated, and he was very thirsty; he dozed for about five minutes on his side, and then turned again on to his back and began talking about his illness, saying he had had frightful paroxysms of pain since he came to England in January, and that he would far rather die than go through so much pain again; but he did not seem to have any sense of impending death. Finding his skin very cold and clammy, and his pulse extremely feeble, a hot bottle was put to his feet, and another blanket thrown over him. The latter process he objected to strongly, and it was removed. About this time (2 A.M.) I took his temperature and found it only 95°; his respirations were not counted, but though shallow they were not laboured, and not so quick as before; there was occasional hiccough, but it passed off in an hour after getting to bed, on sucking ice. He told me his temperature had been raised for three months past, and that the

highest point reached had been $103^{\circ}2$. There was no report of any sudden rigor.

'During the night his thirst was great; he took freely of the iced milk, soda, and brandy, but was afraid of hot beef-tea.

'He insisted on my lying down, saying he thought he could sleep himself; he would not have more covering over him. About 5 A.M. the nurse roused me in the next room and said she thought he had fainted. I felt his pulse. It was fainter than before, and intermittent; he had more brandy, and again dozed (after his temperature had been taken and found to be 94°). About 6 A.M. he cried out that he was blind, but in a second or so he said he could see all right and was quite cheerful; his voice was quite strong. About 7 A.M. Sir J. Fayerer saw him again. At 8 I left him (in the same state as he was an hour earlier), intending to return at twelve o'clock. At eleven o'clock I was told of his death half an hour previously. The nurse informed me that she was sitting beside him as he was dozing quietly, when she heard a slight gurgling in his throat and found he had ceased to breathe.

'Mr. M., æt. 45, died 10.30 A.M., March 6, 1882. Post-mortem examination, 4.30 P.M., March 8. Abscess of liver. Thrombosis of pulmonary artery.

'The body presented no external evidence of disease, and the abdomen was not distended. On making the usual incision, some reddish brown puriform fluid escaped from the lower part of the abdomen. The intestines were pale and not injected, but there were some small patches of recent lymph on the surface of the ileum, and also on the stomach where it came in contact with the liver. Between two and three pints of the thick reddish fluid was removed from the peritoneal cavity, and on extracting the liver a further quantity was expressed. The gall bladder was not distended and was free from gall-stones. The anterior surface of the liver was pale and smooth, the posterior surface of the right lobe containing a large abscess (it had communicated by a small opening with the peritoneal cavity, capable of holding eighteen ounces of water), which was globular in shape. The abscess was solitary and the pus thick and yellow, and the remaining portion of the organ presented nothing noteworthy. The right kidney formed one of the walls of the abscess; the renal structure was healthy and the capsule non-adherent. The left kidney was normal, and the spleen was not enlarged. The stomach was pallid, distended

with gas, and contained some fluid milky matter stained with bile. The small intestine contained much mucus, but was otherwise healthy. There was a curious diverticulum from the ileum about four feet from the ileo-cæcal valve; this pouch was about three inches in length, and rather larger in diameter than the intestine itself. The large intestine was perfectly healthy, with the single exception of one small cicatrix just above the anus. The vermiform appendix contained fæcal matter. The lungs were very pale and emphysematous, and free from adhesions; there was hypostatic congestion of both bases.

'The pericardium contained a considerable amount of serum. The pulmonary artery held a firm thick white clot, which was continued from and nearly filled up the right ventricle and auricle. The left chambers of the heart were empty; all the valves and the structure of the heart were normal.'

CASE XLVIII.

FEBRICULA.

(Medical College Hospital, Calcutta, Dr. McConnell.)

Ram, æt. 50 years, Hindu, admitted September 22, 1881, discharged September 26.

Remarks.—A Bengali servant, attack of fever sudden on the previous day. No complications, except headache and sleeplessness for first two days after admission. Treated by diaphoretics and cinchona alkaloids, with 20 grains pot. bromid. at night. Discharged cured. (Temperature Chart No. 9.)

CASE XLIX.

FEBRICULA.

(Medical College Hospital, Calcutta, Dr. McConnell.)

Maiboob, æt. 18 years, Mahomedan, admitted August 8, 1881, discharged August 15.

Is a boatman on the River Hooghly. Simple febricula with no complications. Treated by diaphoretics and cinchona alkaloids. No rise of temperature after the 10th. (Temperature Chart No. 10.)

CASE L.

SIMPLE CONTINUED FEVER.

(Medical College Hospital, Calcutta, Dr. McConnell.)

A. P., æt. 21, a student-apprentice (military class), states that he has been suffering from fever, more or less continued,

NAME

Ram Hindu

Case 48

Age

50

Sex

Place

Med College Hospital Calcutta

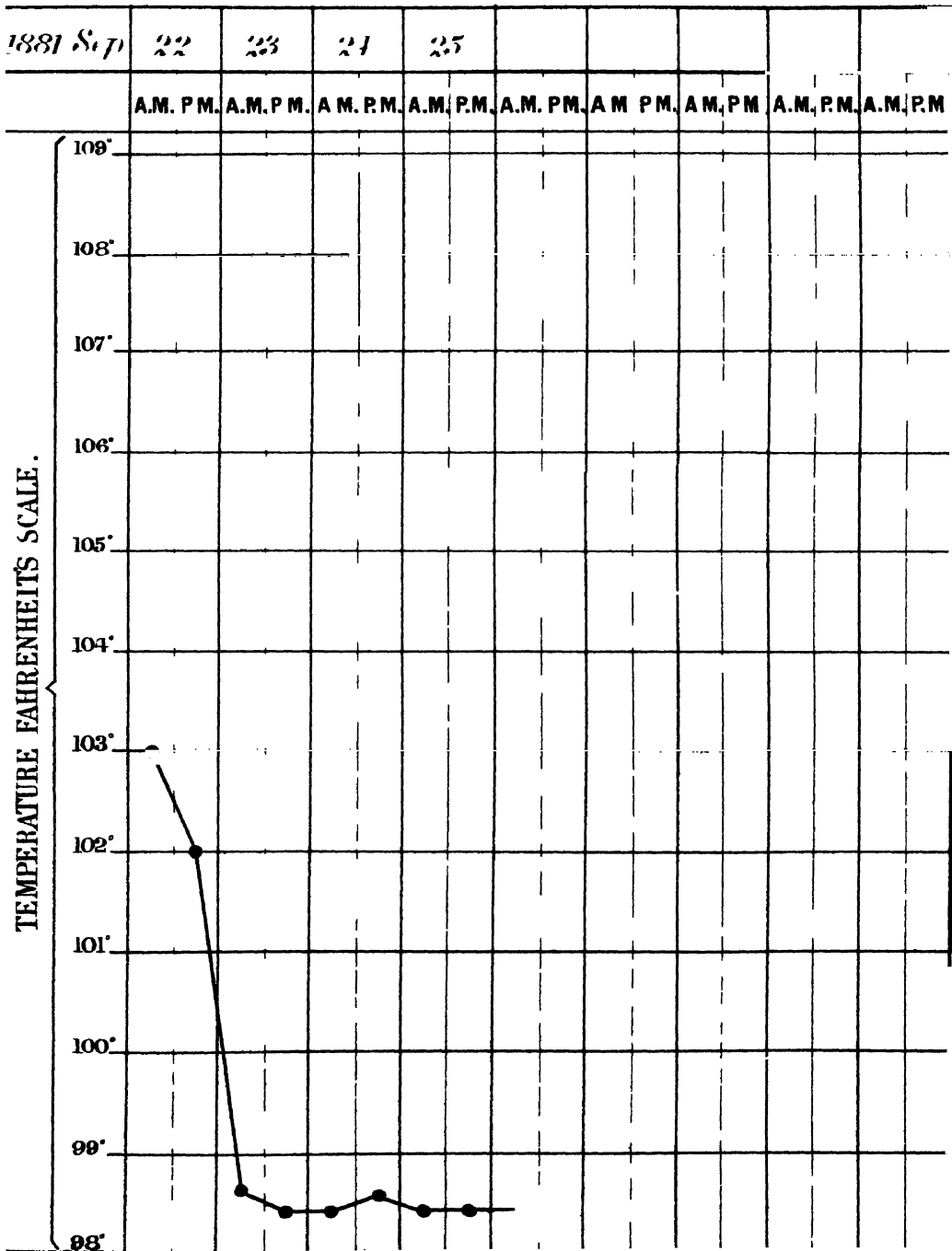
Disease

Fibricula

Medical Officer

D.M. Connell

Result, Cured



for the last four days. Admitted into hospital August 31, 1881. Is a fairly strong and healthy-looking lad. The face is flushed. Skin hot but moist. Pulse quick but regular. Tongue coated. Much headache. Bowels inclined to be loose; the motions are bilious. There is no tenderness over the liver, and no appreciable enlargement of the spleen. Has an irritable, dry cough. The respiratory sounds are normal, except at the bases, posteriorly, where a little dry rhonchus and sibilant cooing is audible.

He was ordered liq. ammon. acet. mist. $\mathfrak{z}\text{j}$ every three hours, a bromide draught at night, and the following powder twice a day :

\mathfrak{R} Sodæ bicarb., gr. v.
Palr. Ipecac., gr. i.
Calomel, gr. i.

The bowels, loose on admission, became much constipated and difficult to move, and the headache with high temperature persisted for over a week. Then gradually the tongue began to clean, and some appetite returned. The alterative powders were omitted on September 6, and the saline mixture with an additional $\mathfrak{z}\text{ii}$ of liq. ammon. acet. was persevered in (every three hours). On September 8, the temperature having fallen, for the first time, to the normal ($98^{\circ}\cdot4$ F.), four grains of quinine with half a grain pulr. digitalis was ordered in the morning, and repeated at noon. Notwithstanding this, the temperature reached $102^{\circ}\cdot2$ F. that evening, but the next morning rapid defervescence with profuse sweating ensued; and the powders being continued, the temperature on the 9th only rose to 99° F., and thenceforth the fever terminated critically. The boy was very much reduced by the attack, and for some days was unable to leave his bed. Cinchona alkaloid (gr. v. in solution) was substituted for the quinine on September 11, and continued up to the date of his discharge, September 19. He has remained quite well ever since.

CASE LI.

SIMPLE CONTINUED FEVER.

(Medical College Hospital, Calcutta, Dr. McConnell.)

E. D., an East Indian child, æt. 7 years, admitted on Aug. 30, 1881. She is said to have been suffering from fever for the last seventeen days. There is no splenic or hepatic enlargement. No eruption on the skin. The abdomen is soft but tender on pressure in the right hypochondrium. Bowels moved once or

twice daily. There is slight cough. Wheezing and cooing sounds are audible at the bases of both lungs. Skin hot (temperature 103°). Pulse quick, but regular. Ordered ℥ss. of liq. ammon. acet. mist. with m℥. vini. ipecac. every three hours, and a sinapism applied to the chest.

By September 2 the cough had all but disappeared. The bowels were moved regularly daily, and the motions were healthy, but the high temperature persisted.

On September 4 she was ordered (in addition to the mixture) the following powder three times a day:—

℞ Hydrarg. subchlor.
Pulr. ipecac.
Pulr. antimonialis, aa. gr. $\frac{1}{2}$.
Soda bicarb. gr. iii.

and warm water sponging (to promote the action of the skin).

On the morning of September 7 the temperature fell to 98°·8 F. The child had slept well and was altogether better.

Cinch. alkaloid, gr. iii.
Calomel, gr. $\frac{1}{2}$.

ordered twice a day and the other powder omitted.

On the 9th, the temperature becoming normal for the first time, she was given cinchona alkaloid (gr. v. in solution).

For two or three days the temperature rose slightly in the afternoon; but on September 14 complete defervescence took place, and she was removed by her parents from the hospital next day. (Temperature Chart No. 11.)

CASE LII.

THERMIC FEVER.

(General Hospital, Madras, Dr. Sturmer.)

J. C., æt. 57, European, a carpenter, admitted May 20, 1881.

History.—Has had fever for the last three days. About a month ago he suddenly fell down while working in the sun, and lost consciousness for twenty-four hours. He resumed work, but has not felt well since. About a week ago he vomited and continued to be sick for two days. His eyesight then became affected, and everything appeared dim. This improved on resting. On the 19th he had a severe attack of fever, which still continues; he attributes it to working in the sun. Heart regular. Tongue coated with yellow fur. Appetite impaired. Com-

plaints of severe headache, chiefly situated at the back of the head. Pupils contracted; is dull and semi-delirious. Sleeps badly. Skin warm and moist; body covered with prickly heat. Scanty urine, and of high colour. Quinine gr. x., bis die.

21st.—Bowels moved after aperient. Cold to the head, and tepid bath if temperature rises above 103°. Quin. sulph. gr. xxv., statim.

22nd.—Restless last night, bowels acted once. Quin. sulph. gr. xx., statim.

23rd.—Patient was slightly delirious last night; slept badly. Tongue coated with yellow fur. Motions scanty, contain scybala. Mist. sennæ co. ʒj, statim. Died at 4.20 P.M. Had a bath at 1 P.M., when the temperature was 108°·6; it fell then to 105°.

Post-mortem, 8.45 A.M. May 24.—Body that of a large, well-built man. Height, 6 feet; weight, 164 lbs. No marks of syphilis. Rigor mortis in lower extremities only.

Head.—Slight congestion of dura mater superiorly, otherwise healthy. Texture of brain firm, not congested. Puncta cruenta not increased; small amount of pale clear fluid in lateral ventricles. Choroid plexuses pale, not distended; in both there is a cluster of small serous cysts. Vessels appear normal.

Thorax.—Lungs do not collapse, and almost entirely conceal the pericardium. No fluid or adhesions in either pleura. Pericardium and heart are coated with a thick layer of fat. About 6 drachms of reddish-yellow clear serum in pericardium. Tricuspid orifice admits four fingers; cavities are large, but preserve their relative proportions; valves healthy. Right cavities contain considerable partly discoloured clots, and left some black fluid blood and black soft clots. Heart flaccid, weight without clots, 15 oz. Patch of atheroma in aorta. Muscular tissue of heart pale and flabby. Right lung, crepitant throughout, congested posteriorly, weight 30 oz.; left, same state, 29 oz.

Abdomen.—Spleen enlarged and so soft that it breaks down in removing it, weight 28 oz. Capsule thin and easily ruptured. Left kidney weighs 7 oz.; posteriorly there is a large cyst, about the size of a goose's egg, containing clear serous fluid, and enclosed between layers of capsule; kidney tissue normal. No reaction with iodine. Liver weighs 68 oz., somewhat soft and flabby. Lobules rather indistinct; tissue friable. No reaction with iodine. Gall bladder contains some healthy as well as some inspissated bile. Bladder contracted. Intestines not examined. (Temperature Chart No. 12.).

CASE LIII.

THERMIC FEVER RESULTING IN PARTIAL PARALYSIS.—RECOVERY.

G. C., æt. 44 years, military officer, service in India twenty-seven years.

Case as reported from India.

This officer had sunstroke in 1856, and ever since cannot remain long in the sun without his head being more or less affected. He had sick leave to England in 1858 on account of wounds, and two months' sick leave in India in 1872, when he suffered from very severe attacks of fever. With the above exceptions he enjoyed very good health until he went on active service with his regiment, and was stationed at Ali Musjid, where he was much exposed to the sun.

On May 15 he felt slight numbness and loss of power in his right arm, and later in the day the left arm and legs became similarly affected; gradual and steady increase of this loss of power took place, till on May 20 he was unable to stand or walk. This was accompanied by pain in the loins and shoulders. On May 21 he was sent to Peshawur with the view of obtaining sick leave. The Board declared him unfit to travel, and recommended that he should be treated in Peshawur. On May 24 he was rather pallid; conjunctivæ were slightly injected, and there was slight impairment of vision; pupils were regular and responded to light; he was unable to close his eyelids (especially the left) properly, and the tears trickled down his cheeks; the left side of his face was flattened and smooth, he spoke slowly and was unable to articulate labial sounds, to whistle or puff out the cheeks. There was neither paralysis of tongue nor palate, nor loss of taste, nor was the sensibility of the lips impaired. There was complete paralysis of the arms and hands, the fingers were in a flexed position, and he was unable to straighten them; there was a tingling sensation in the forearms and hands, but sensation was only slightly impaired, and if his fingers were touched he felt it. There was complete paralysis of the legs, whilst sensation in them was the same as in the hands. There was neither headache nor vertigo, and his intellect had been unaffected throughout. Heart sounds were healthy, though the action was rather weak; the pulse was small, compressible, and rather quick. No tenderness over any part of the spine could be discovered on

the application of a hot or cold sponge over it. He complained of a tightness across the chest, with difficulty of breathing.

Condition when sent home.—The facial paralysis has almost disappeared. Though there is still a slight flattening of the left side of the face, he can close his eyelids perfectly, speak with ease, bring his lips together, puff out his cheeks and whistle.

Power of motion in the hands and arms has returned to a great extent; he can grasp his handkerchief and wipe his face, put his hand to the top or back of his head, and put on and take off his spectacles; his fingers are, however, still rather cramped, and he suffers a good deal of pain in them. Motion and sensibility in the legs are improved, and though he cannot raise them from the ground he can draw them up while in a recumbent posture. He, however, suffers a good deal of pain in them. His general health is good.

Treatment.—Iodide and bromide of potash, iron and tonics.

This officer had recovered, in England, in December 1881, and was about shortly to return to India.

The following is his statement of his case (in continuation of the medical one):—‘I think it was on Saturday, May 22, 1880, I lost entire use of my hands and arms. On the following Tuesday, the 25th of that month, I began to recover use of them, was able to move my hands up to my face, and about three weeks later to hold a paper and feed myself; but it was not before the end of June or the first week in July I could draw my feet up—that is, bend my knees. On July 10 I left Peshawur for Calcutta, where I arrived on the 17th, so much better that I could bend my knees with ease. Unfortunately no one told me of the necessity of straightening my legs occasionally and working the muscles under my knees, and I had great difficulty in getting my legs straight in consequence of having kept them drawn up for three weeks. My appetite throughout was good. I improved rapidly from the good food I got in Calcutta and on board ship. It was on the 25th or so of August I managed to draw myself up from my bed by the aid of a chair, and stand for a few seconds. I walked twice that day about ten steps, supported by a couple of men. Each day I walked more, till on September 5, the day I landed, I could walk without assistance about 100 yards. By the end of November 1880 I walked a mile, and until I injured my muscles by drawing a mowing machine which was out of order, I never had an ache or pain. Since that time, last June, I have been subject to cramps after over-exertion. My health in

every other respect until November 9 was excellent. On that day I had some sandwiches at a restaurant, which must have had bad meat in them, for I was violently sick and purged a few hours later, and had the worst attack of cramps in my legs I have ever had. I am nearly all right again, but not so strong or able to walk so much as before that attack. I hope care and a moderate amount of exercise will gradually completely restore me.

‘I am recommended to take burgundy and “Easton’s Syrup.” I take a bottle of this, and when it is finished stop for a few days, and then go on with it again. In fact nothing could have been better than my health had been until I took that lunch; the sickness brought on indigestion; even that has now passed away, and I am again making rapid strides to good health. In the summer I thought myself stronger than I really was, and did too much, which brought on my only complaint—cramp in my legs. I was taken to see Dr. B., who after a long examination pronounced me to be perfectly recovered from the illness, but that it would take time and care to restore me completely. I think I have told you all stages of my progress to recovery.’

CASE LIV.

NERVOUS PROSTRATION, THE RESULT OF THERMIC AND MALARIAL FEVER.

M. S., æt. about 30. (Case reported from India.) During service throughout the Mutiny his health was very good, and remained so up to the year 1868.

In July of 1868, while engaged in police duties near the Terai, he had sunstroke, and was prostrated for some months, which necessitated his being sent to the hills. In September 1869, after being on duty in the Sunderbuns, the Civil Surgeon again sent him on leave, as the effects of the sunstroke still troubled him, and he had been suffering in the interim from fever and dysentery contracted in the Sunderbun jungles. In 1870 he was appointed to British Burmah in the Forest Department, and went out into the forests in May 1871.

He remained at forest work till 1873, and fairly broke down from the effects of heat, exposure, and repeated attacks of fever and dysentery, and had to go to sea to recruit. In the beginning of 1874 he was sent to Rangoon, and after being there for a year, and being still ill, he obtained leave to England.

In April 1877 he returned to India, and was immediately placed on forest duties, which involved exposure to the sun all day from sunrise to sunset. February 1878 he was relieved from duty, being thoroughly prostrated. He was recommended to leave the country at once on the following grounds:—

‘I have found him suffering from nervous and other symptoms which have been undoubtedly produced by prolonged exposure to the sun. He has suffered (he states) twice previously in a similar way. His present employment entails exposure to the sun almost the whole day; he is quite incapable of performing such duties without risk.’

Having but recently returned from England the patient was determined to try and do his work, and was again placed on forest duty, but in April 1879 was compelled again to avail himself of leave to England.

This officer is so completely prostrated, and his nervous system so depressed, that he will not return to India.

CASE LV.

THERMIC FEVER.

(Medical College Hospital, Calcutta, Dr. McConnell)

J. R., æt. 40, a groom employed in a livery stable, was admitted into hospital on May 22, 1880, with a suppurating hydrocele. This was treated by incision and dressed. In a few days the wound had healed, and the man appeared to be progressing quite favourably. He was, indeed, about to be discharged, when on June 12, a very hot afternoon, he was found (at 4 P.M.) in bed semi-comatose; the breathing laboured and sighing, the skin pungently hot, and the temperature 104° . Cold douching, ice to head and neck, and a 10-grain hypodermic injection of quinine were administered, but notwithstanding this the temperature rose by 10.30 P.M. to $108^{\circ}4$. Another injection of quinine was given, but had no appreciable effect, and he died at 11.30 P.M. *Post-mortem examination* ten hours after death. Body well nourished. Rigor mortis strong, especially in the lower extremities. Pupils a little dilated.

The wound in the scrotum has healed; only a line or cicatrix about two inches in length can be seen. Beneath it, the subcutaneous tissues are healthy, and the sac of the tunica vaginalis is found filled with a considerable amount of soft, jelly-like

material, evidently organising lymph. The testicle and cord are quite healthy.

Head.—The sinuses of the dura mater are loaded with dark fluid blood. The vessels of the pia mater intensely engorged, and in the meshes of this membrane, over the upper surface of both cerebral hemispheres, a large amount of serous effusion is found. The brain substance is moderately firm, a little hyperæmic. The lateral and third ventricles are filled with dark pinkish serum. The central ganglia, medulla, and cerebellum are all healthy. The vessels at the base of the brain are filled with dark fluid blood.

Lungs.—Large and heavy. Intensely congested. Dark purplish in colour. Exudation on pressure and incision of a great quantity of fluid dark blood and frothy sanguineous serum. Consistency soft and œdematous.

Heart.—Pericardial cavity contains an ounce of dark straw-coloured serum. Heart somewhat large, external surface coated with a good deal of yellow fat. Left ventricle feebly contracted. Right cavities loaded with dark fluid blood; in the auricle, in addition, a soft, shreddy, dark clot. A small quantity of fluid dark blood in the left auricle and ventricle. The valves and endocardium healthy. All the abdominal organs displayed dark venous congestion, but nothing else specially remarkable.

This is an interesting case, as showing that heat apoplexy is not always due to exposure to the direct or even indirect rays of the sun, and that therefore the term 'insolatio' is one not uniformly applicable to the affection. It is by no means an infrequent occurrence among European soldiers, even while confined to their barracks in the hot months of April, May, and June. I give the case merely as illustrative of a well-known fact.

CASE LVI.

THERMIC FEVER.

(Medical College Hospital, Calcutta, Dr. McConnell.)

J. B., an English seaman, æt. 34, lodging in Bow Bazar Street, was admitted into hospital on the evening of June 1, 1878. He gives a history of exposure to the sun during the past two days, but denies having indulged in liquor too freely. Complains of severe headache, which set in suddenly at 3 P.M. (this afternoon). The face is flushed. The skin pungently hot and dry. Temperature now (6 P.M.) 107°·2. Tongue dry and

parched. Pulse small and weak. The patient gradually passed into a comatose condition, with stertorous breathing and lividity of the face and neck. Cold douching, ice to head and neck, packing in wet sheets, &c., were carried out vigorously, also a cathartic enema given, and 10 grains of quinine administered hypodermically. After the hypodermic injection the temperature fell to $102^{\circ}6$; but again rose rapidly, and a few minutes before death the thermometer in the axilla registered 108° . Death took place at 8 P.M. (two hours after admission), and was preceded by a kind of convulsive fit, ending in rigidity of all the limbs, and rapid, wide dilatation of the pupils.

Post-mortem examination twelve and a half hours after death. Body well nourished and muscular; pupils dilated. Rigor mortis well marked in the lower extremities, very slight in the upper. Patches of post-mortem discoloration (mottling) are observed over the skin of the abdomen, and a dusky purplish colour of the surface along all the dependent portions of the body (back, back of neck, back of thighs and legs, &c.).

Head.—Sinuses of dura mater filled with dark fluid blood. Vessels of pia mater intensely engorged. No serous effusion. Brain substance slightly hyperæmic, of about normal consistency. Half a drachm of serous fluid in each lateral ventricle.

Heart.—Large. Feebly contracted. Right cavities loaded with a dark fluid blood, and a small quantity of the same in the left chambers; no coagula. The valves and endocardium on the right side are healthy; on the left the mitral and aortic valves (especially the latter), and also the lining membrane of the aorta, are considerably thickened, hard, opaque, atheromatous.

Lungs.—Large. Darkly congested, and loaded with frothy, sanguinolent fluid.

Abdominal organs generally show great venous congestion, but nothing else specially remarkable.

CASE LVII.

ARDENT OR THERMIC FEVER.

(Medical College Hospital, Calcutta, Dr. McConnell.)

W. F., æt. 33, a European seaman, was admitted into the Medical College Hospital on March 26, 1875. He stated that he had been suffering from 'fever and ague' for the last ten days.

Is a strong, well-built man. *The countenance is flushed. The*

tongue moist, but much coated. Complains of headache. The skin is hot and dry, temperature 104° . Pulse full, hard, excited, 108. There is tenderness on pressure over the hepatic region. No splenic enlargement. No cough.

March 27.—Temperature $105^{\circ}4$. Has passed two or three loose feculent and bilious stools during the night, and vomited once (also bilious fluid). The conjunctivæ are injected. Severe headache. Pupils normal. Evening temperature $105^{\circ}4$ F.

March 28.—Temperature $102^{\circ}6$. Mind wanders occasionally. Has to be roused to answer questions. Tongue dry. No difficulty of breathing or in swallowing. Evening temperature $104^{\circ}8$. Remains in the same condition.

March 29.—Is quite delirious and evidently sinking. Temperature 107° . Died at 11.30 A.M.

Treatment.—Cathartic enema. Ten leeches to the temples. Sinapism to the right hypochondrium. Fever (diaphoretic) ixture with *mv vini. ipecac.* and quinine in small doses.

Post-mortem examination twenty-two hours after death. Body well nourished and muscular. Rigor mortis strong. Pupils dilated. The integument of the neck, ears, and posterior surface of the body generally has a livid dark purple colour. Dura mater darkly blood stained. On its section, about two ounces of sanguineous serum escapes. The vessels of the pia mater are greatly injected. The brain substance throughout is abnormally vascular, the puncta large and bleeding freely. The lateral ventricles contain a few drops of bloody serum. Both lungs intensely congested and dark, slightly softened and cedematous consistency.

Heart.—Large and quite flaccid. Right cavities filled with fluid blood, no coagula. Left chambers almost empty. Valves, &c., healthy. Weight of heart 11 ounces.

Liver.—Substance moderately firm, of dark red colour; the surface presents a few sanguineous blotchings. Large portal and hepatic veins loaded with fluid dark blood. Lobular structure ill defined, and preternaturally hyperæmic. Gall bladder half-full of bile, thin, of a turmeric-yellow colour, measuring out 3 drachms. Weight of liver 3 lbs. $4\frac{1}{2}$ oz.

Spleen.—A little enlarged. Substance exceedingly soft, lumpy, and dark. Proper structure quite indistinguishable. Weight 7 oz.

Kidneys.—Large, dark, and heavy (venous congestion). Weight of right 5 oz., weight of left 8 oz.

Mucous membrane of stomach corrugated, thick, dark-purplish in colour. That of the middle intestines is for the most part pale, but in patches, irregularly bile-stained. The cæcum shows recent vascularity and bile-staining, rest of large gut healthy. Mesenteric glands slightly hyperæmic. (Temperature Chart No. 13.)

CASE LVIII.

SIMPLE CONTINUED FEVER.

(Under the care of Dr. Joubert.)

Dr. Joubert, of the General Hospital of Calcutta, says: 'I have for the last year or two (or more) met with a fair number of cases of continued fever, to my mind quite distinct from enteric, which lasted exactly twenty-one days, when complete convalescence occurred, and in which quinine appeared to have absolutely no effect. So marked are these cases that after a few days' observation defervescence can be predicted, on, and not before, the twenty-first day, and usually occurs. There is very rarely delirium, if ever; never the formation of sordes on the teeth and tongue, no decided abdominal pain, sometimes gurgling on pressure if there be looseness of the bowels—a symptom not often present; no rose spots, and not that marked emaciation and loss of muscular strength, and long-continued debility, so very common after mild cases even of true enteric fever. These cases are often called "typhoid fever," but I am not inclined to agree to this designation.'

E. H., æt. 23 years, ship's steward, three months in India, European. Admitted General Hospital, Calcutta, June 15, 1878;¹ discharged June 23.

Suffered from fever and headache for five days before admission, the effects of heat upon board ship. Face flushed; pupils dilated; skin hot and dry; tongue furred; pulse full and frequent. Pain all over body; bowels open; 16th and 17th, symptoms unchanged (he had several cold baths). Pupils semi-dilated; conjunctivæ injected. 18th, better; 19th, improving; 20th, face flushed; pulse full and frequent; tongue slightly furred and fissured; bowels open after purgative; skin hot and dry; 21st, skin cool and moist; tongue clean; constipation; no headache or muscular pain; 22nd, convalescent. The only treatment in this case was cold-water baths twice or three times daily for three days, and diaphoretic mixture.

¹ The heat was very great in June 1878.

CASE LIX.

SIMPLE CONTINUED FEVER.

(Under the care of Dr. Joubert.)

H. A. M., æt. 32, chief ship's officer, American, six months in India. Admitted General Hospital, Calcutta, June 17, 1878; discharged June 27, 1878.

Taken ill two days before admission with fever. Has pains in the back; tongue furred and brown; skin hot and dry; pulse quick and full; bowels freely open. 18th, face flushed; conjunctivæ injected; pupils normal; tongue thickly coated white; headache; pains all over; pulse full and quiet; skin hot and dry; thirst; bowels open. 19th and 20th, the symptoms continued about the same; there was slight delirium occasionally, and the bowels were loose. The patient had very frequent cold baths, which lowered the temperature one or two degrees for a few hours at a time only. After last bath, on evening of 20th, temperature fell to $102^{\circ} 4$, and the next day to normal, never rising again. Convalescence complete on the 22nd.

Treatment.—Cold baths and a saline diaphoretic, and on 20th, 10 grains of quinine twice.

CASE LX.

REMITTENT FEVER WITH ENTERIC SYMPTOMS.

(Under the care of Dr. Nicholson.)

R. M., æt. 22 years, no occupation, European, one year in India. Admitted General Hospital, Calcutta, April 17, 1877; died May 1.

On admission complained of having had headache for eight days, with constipation. Tongue furred, and fever present. Noted as 'better' on 18th, 19th, 20th, and 21st. Looseness and fever on evenings of 21st, 22nd, 23rd, 24th, and 25th, looseness and high fever, and tenderness on pressure in iliac fossa; 26th, no change; patient drowsy; 27th, drowsy and flushed; tongue coated at centre, edges clean; bowels loose; 28th, condition unchanged; 29th, bowels not so loose; tongue furred and dry, edges red and raw; vomiting; gurgling in right iliac fossa; delirium in evening; 30th, delirium all night; drowsy; bowels moved once in bedclothes; May 1st, delirium continued; motions passing in the bedclothes; death at 11.20 P.M.

Post-mortem Notes.—The mucous membrane of the lower three feet of the ileum presented the following appearances:—Several Peyer's patches, swollen and deeply red in colour. In the cæcum a small patch of thickened mucous membrane, about four inches in length, and upon it several minute ulcers. Large intestine healthy throughout; spleen enlarged; other organs healthy.

CASE LXI.

ENTERIC FEVER.

(Medical College Hospital.)

Gopal, a Hindu child, æt. 8 years, was admitted into hospital on Dec. 30, 1873. The mother, an ignorant native woman, could give no very clear history of the case; but as far as could be ascertained, it appeared that the boy had been suffering from fever for about a week, that it was more or less *continuous*, and that for the last two days he has been delirious.

The child is much emaciated. There are black sordes on the teeth and lips. Pulse exceedingly feeble and small, 112 per minute; respiration hurried, 36 per minute. He is delirious, and there is a good deal of restlessness, and subcaltus tendinum. The abdomen is flat, and there is uneasiness evidenced on pressure over the cæcum and round the umbilicus. Tongue dry and fissured. No spots or any kind of eruption over any part of the body.

Dec. 31. Seems a little better to-day. Is able to answer questions. Temperature 100°. Tongue still dry. Has had one stool—'formed,' but deficient in bile (muddy-coloured). Evening temperature 100°.

Jan. 1, 1874. Temperature 101°. One stool, thinner and very offensive. Very restless during the night, and at times talked incoherently. Evening temperature 102°.

Jan. 2. Two stools during the night of the same character as before, still deficient in bile, and with them were passed three round worms. Tongue still dry. Restless and sleepless most of the night. Evening temperature 103°.

Jan. 3, 4, 5, and 6. No improvement; during the whole of the 6th he was wildly delirious, and refused nourishment and medicines.

On January 7 the body became bathed with perspiration; the pulse flickering and almost imperceptible at times, and he died from exhaustion next afternoon.

Treatment.—At first bark and ammonia, then quinine and dilute nitro-muriatic acid, turpentine stupes to the abdomen, milk, soup, and latterly port wine.

Post-mortem Examination (22 hours after death).—Body emaciated; rigor mortis present in lower extremities, none in the upper. No spots or eruption.

Brain and membranes healthy, the former rather anæmic. *Lungs*: just beneath the visceral pleura along the posterior surfaces of both organs, were large dark ecchymoses and ecchymotic patches; and on section were seen similar scattered spots of blood-staining and extravasation, contrasting with the otherwise anæmic appearance of the general pulmonary structure. Crepitation and sponginess normal.

Heart.—Half an ounce of straw-coloured fluid in pericardium; heart moderately contracted; right cavities contain only a little thin-looking, dark (claret-coloured) fluid blood. The left cavities are empty; valves and endocardium healthy; muscular tissue of heart somewhat paler and softer than normal.

Abdomen.—Peritoneum healthy.

Liver.—Large; capsule thickened and adherent (old adhesions) to the diaphragm; both the surface and interior of the organ present a canary yellow colour from almost uniform bile-staining; consistency very soft; lobular structure ill-defined. Gall-bladder contains about 6 drachms of clear, transparent, amber-coloured bile; weight of liver, 33 oz.

Spleen.—Enlarged; capsule opaque and thickened; substance soft and of an intense dark-purple colour: weight 8 oz.

Kidneys.—Healthy.

Stomach.—Mucous membrane corrugated and pale. That of the *small intestine* presents nothing remarkable until the lower half of the ileum is reached. In this portion there is found much general and recent vascularity of the mucous surface, and the glandular structures (solitary follicles and patches of Peyer) are throughout enlarged, prominent, and ulcerated. The ulcers increase in size and number as the ileo-cæcal valve is approached, and the last 30 inches of the ileum are extremely well marked. They are round or oval, and strictly confined to the limits of the glandular structures. Six inches above the valve there is one quite 2" in length by 1" in breadth, implicating the whole of a Peyer's patch; another 1" \times $\frac{1}{2}$ " about four inches below this; a third $\frac{1}{2}$ " \times $\frac{1}{2}$ " just above the valve; and one about the size of a rupee (florin) involves the iliac surface of the valve itself. They

all penetrate deeply, the largest reaching the peritoneal coat, which is much thinned. Each ulcer is surrounded by a dark purplish zone of congested vessels. In several the sloughs have not yet separated, or only partially so.

The mucous membrane of the *large intestine* is pale, and its glandular structures are unaffected.

The *mesenteric glands* are all enlarged, some to the size of half a walnut; are soft and intensely hyperæmic on section.

The stomach contains about 4 oz. of thin, reddish fluid, with shreddy mucus. About 3 oz. of greenish-yellow, thin, feculent fluid is found in the small intestine, and a small quantity of formed muddy-coloured fæcal matter in the large gut. (Temperature Chart No. 14.)

CASE LXII.

ENTERIC FEVER.

Badam Behari, a Hindu, æt. about 35, a 'bearer' by occupation, was admitted on Sept. 16, 1875, into Medical College Hospital, and died the next day.

His history was that for the last 20 or 25 days he had been suffering from fever of a continued type, but had not been under any kind of treatment. The bowels had been throughout constipated rather than loose. Is weak and emaciated; pupils normal; conjunctivæ somewhat suffused and injected; tongue, dry, hard, and devoid of epithelium; dark sordes over lips and teeth; skin, hot and dry; temperature $101^{\circ}2$; no rose spots or any eruption on the skin; pulse small, weak and frequent, 114; abdomen sunken; slight tenderness over the cæcum; a few moist râles at bases of both lungs, and comparative dullness at the right base; no enlargement of spleen or liver perceptible. Is quite conscious. Evening temperature 101° ; pulse 108.

Sept. 17.—Passed one stool during the night. It was loose, feculent, and bilious (no mucus or blood). Is weaker, and talks incoherently; temperature 101° ; pulse 105. In the afternoon the temperature fell to $97^{\circ}8$ (sub-normal); the extremities became cold, and the patient died in a semi-comatose condition.

Post-mortem Examination ($16\frac{1}{2}$ hours after death).—Rigor mortis strong in the lower extremities, none in the upper; conjunctivæ jaundiced; pupils dilated; no spots or eruption.

Brain and membranes.—Darkly congested, but no inflammatory effusion.

Lungs.—Dark and codematous, especially along the posterior margins and at the bases.

Heart.—Pretty firmly contracted; right cavities contain fluid dark blood, and soft, very slightly decolorised clots; left auricle empty; left ventricle holds a soft dark clot; valves and endocardium healthy.

Peritoneum healthy.

Liver.—Of about normal size; substance soft, pale reddish-brown; lobular structure almost indistinguishable; bile ducts prominent and full; gall bladder contains about $\frac{1}{2}$ oz. of thick, inspissated bile; weight of liver, 42 oz.

Spleen.—Enlarged and heavy; substance very soft; almost diffuent, and very dark; weighs 16 oz.

Kidneys.—Both rather large, heavy and dark, otherwise normal.

Stomach.—Mucous membrane slightly corrugated, somewhat thinned, and shows patches of dark-purple congestion and ecchymosis along the lesser curvature. The mucous membrane of the *small intestine* is a good deal bile-stained in the duodenum and jejunum. That of the whole of the ileum is intensely congested, and the glandular structures are ulcerated. These ulcerations occupy the long diameter of the gut, opposite to the attachment of the mesentery, and involve both the solitary follicles and patches of Peyer. They increase in number and size as the ileo-cæcal valve is reached, and are very thickly distributed in the lower third of the ileum. Here they present prominent fungus-like characters; their margins raised, dark-purple, highly injected. Their surfaces irregularly ulcerated, and some are still covered by small adherent yellowish sloughs. Together with such ulceration, prominence and infarction of the same glandular structures exist. Just above the ileo-cæcal valve the ulceration is extensive and of irregular outline.

In the *large intestine* there are patches of vascularity here and there, no uniform congestion, and no ulceration.

The *mesenteric glands* are enlarged, some to the size of filberts; and on section, dark-purple, soft, highly vascular.

The stomach contains about 2 oz. of thin, watery, bilious fluid. About a pint of greenish-yellow, thin, fæcal matter is found in the small intestine; and a couple of ounces of thin, muddy-coloured, offensive, fæcal fluid in the large gut.

CASE LXIII.

ENTERIC FEVER.

(Under the care of Dr. O'C. Raye, General Hospital, Calcutta.)

C. V., æt. 27 years, seaman, B.S. 'Eurydice,' English, six weeks in India. Admitted on May 12, 1877; died May 24, 1877.

Had been working in the sun for several days, and complained of weakness, giddiness, and headache.

The notes up to the 17th indicate symptoms of ordinary continued fever, but on the 17th the pulse is noted as intermittent, and weakness considerable. Bowels loose on 13th, but constipated afterwards. On the 18th delirium and involuntary motions; subsultus tendinum, face flushed. 19th, sordes on tongue, drowsy, pupils contracted, slept well, no delirium. 20th, drowsy, motions in bedclothes, loose and watery, tongue tremulous and covered with sordes, jactitation of limbs, copious perspiration, distinct gurgling in iliac fossa (right). 21st, no delirium, slept; tongue and teeth covered with sordes, involuntary motions, base of right lung dull; hepatic dullness increased, pain on percussion, spleen enlarged. Some doubtful spots on abdomen. Gurgling in right iliac fossa. 22nd, no change. 23rd, delirium, semi-conscious, very weak, picking at bedclothes, other symptoms as before. 24th, died at 8 A.M.

Post-mortem Examination.—A number of thickened ulcerated solitary glands and Peyer's patches in lower third of ileum. Eight or more round ulcers on the ileo-cæcal valve. Mesenteric glands enlarged. Cæcum and colon normal, but pigmented. Base of right lung solidified. Spleen enlarged, weight 1 lb. Other organs normal. (Temperature Chart No. 15.)

CASE LXIV.

ENTERIC FEVER.

(Under the care of Dr. Nicholson, General Hospital, Calcutta.)

C. B., æt. 20 years, ship apprentice, European, 7½ weeks in India. Admitted on August 20, 1877; died August 24, 1877.

History for twelve days before admission, intermittent type, coming on daily. On admission, pulse weak and frequent, skin hot, tongue coated yellow, very weak, bowels open. Partly unconscious that night. 21st, bowels freely open during night, pain in abdomen, gurgling in iliac fossa, no spots. Freely

purged, and sick during the day. No notes till the 24th, when the following :—Very restless; subsultus tendinum, delirium, no motions, free diaphoresis, bronchial catarrh, tympanitis, respiration diaphragmatic. Treatment: Diaphoretics, large doses of quinine, and chalk mixture. Death at 4 p.m.

Extract from Post-mortem Notes.—Ulceration of Peyer's patches in the lower two feet of the ileum, above which solitary glands and Peyer's patches thickened. Large, irregular, sloughy ulcer in cæcum. Solitary glands in large intestine enlarged and thickened. Spleen enlarged. Other organs healthy.

[This would appear to have been of climatic origin.—J. F.]

CASE LXV.

ENTERIC FEVER.

(Under the care of Dr. Raye, General Hospital, Calcutta.)

J. O., æt. 20 years, seaman, B.S., European. Admitted June 16, 1877; discharged September 5, 1877.

This man had apparently been ill for ten days before admission, with headache, thirst, and loss of appetite. Had diarrhœa on admission; furred tremulous tongue; weak, quick pulse, and great debility. There was occasional diarrhœa during the first few days, and pain and gurgling in the right iliac fossa. Some bronchitic râles towards the end of June. Solid formed motions were passed on and after June 30. Some looseness again about July 20 and part of August. The tongue seems never to have been coated with sordes.

The patient appears to have had chicken diet from July 13 to the 20th, when it was reduced to milk diet. Chicken diet was resumed on August 10 and continued till discharge on September 5. The case seems to have been a doubtful one of true enteric fever, for at no period do severe 'typhoid' symptoms appear to have been present.

CASE LXVI.

ENTERIC FEVER.

(Under the care of Dr. Nicholson, General Hospital, Calcutta.)

J. M'D., æt. 31 years, chief officer of ship, European, a month and a half in India. Admitted February 5, 1878; died February 13, 1878.

On admission was said to have had fever, with evening

On admission, temperature $105^{\circ}2$, skin moist; tongue furred in centre, red at edges, headache and nausea, and bowels confined. 5th, sordes on teeth and tongue, latter dry and hard; tenderness and gurgling in right iliac fossa; three stools. 6th, drowsy; face flushed; slept badly; spleen and liver not enlarged; other conditions as before; three stools. 7th, conditions the same, but feels better. 8th, some delirium, but slept better; other conditions the same. 9th and 10th, no change, same notes, delirium at night, and death at 6 A.M. of 11th.

Post-mortem Notes.—In the lower third of the ileum, increasing in number from above downwards, were many thickened solitary glands, some covered with a firm slough, others having lost it and presenting an excavated ulcer; quite a mass of them just above ileo-cæcal valve. One swollen Peyer's patch only, covered with yellow lymph. Mesenteric glands enlarged; spleen enlarged, weight 10 oz. All other organs normal. (Temperature Chart No. 17.)

CASE LXX.

ENTERIC FEVER.

(Under the care of Dr. Joubert, General Hospital, Calcutta.)

W. G., æt. 16 years, ship apprentice, English, $3\frac{1}{2}$ months in India. Admitted June 10, 1880; died June 13, 1880.

Reported on admission to have been suffering from fever of an intermittent type coming on every other day for four weeks past. Bowels have been loose all the time. On admission, pulse 108, full and frequent; somewhat delirious, tongue dry. 11th, very delirious all night; bowels loose; skin hot; tongue dry; no pain on deep pressure over right iliac fossa. 12th, delirious all night; one stool only; tongue dry and coated; lungs clear but breathing harsh. Pulse 120; drowsy; skin moist. 13th, passing stools in bedclothes, very watery; tongue as before, less delirious; pulse very weak; no pain on pressure on right iliac fossa; became very delirious during the day, and died at 9 P.M.

Extract from Post-mortem Notes.—In the lower two feet of the ileum were found several scattered thickened and inflamed Peyer's patches, with small firm sloughs stained with fæces on several of them. There were several close together just above the ileo-cæcal valve, but only one of them presented a slough. In the cæcum, ascending and transverse colon, were numerous inflamed solitary glands, about fifty perhaps, many of which

presented a firm faeces-stained central slough, scraped off with difficulty, leaving beneath a depressed ulcerated surface. These enlarged glands were of the size of peas. The rest of alimentary canal healthy. Spleen much enlarged from recent congestion, very dark and friable. Lung quite healthy, also heart, liver, and kidneys.

CASE LXXI.

ENTERIC FEVER.

(Dr. Wisc, Dacca.)

Attended with intermission on the 6th, and by collapse on the tenth day. Death from pneumonia on the fourteenth day of fever.

Jehálman, a Nipálese, æt. 20, an inmate of the Lunatic Asylum, and suffering from melancholia, was a patient in hospital on May 9, 1872, when he was attacked with fever. It was referred to worms, and after a dose of santonine three lumbrici were expelled.

At sunrise on May 11 his pulse was 112, and respiration 24. His tongue was covered with a thin white fur, but the edges and tip were red. A few moist râles were audible at the base of the right lung. He vomited twice, but had no motion.

He passed a restless night, and in the morning the pulse was 112. The papillæ towards the tip of the tongue were red and prominent. The bowels were still constipated. After a careful examination no pneumonia could be detected. In the evening the temperature had fallen, although no quinine had been given. The pulse was still 112, the respiration 24.

The fever continued unabated all night, but on the morning of the 13th a slight fall of temperature occurred. The tongue was dry, red, and cracked. An expression of pain was elicited when pressure was made over the cæcum. He lay sunk in bed, with his legs drawn up. Dullness of the right lung up to the level of the angle of the scapula was very distinct. Vesicular breathing was inaudible, and a few fine crepitations were detected. The pulse was 116, respiration 28. During the night he passed two stools. The urine was pale yellow, sp. gr. 1.014, with slight traces of albumen.

During this day, the fifth of the fever, there was no intermission, and the evening temperature reached 104°·4, the highest point recorded throughout the illness. The pulse was 122, respiration 44. Three thin bilious stools were passed to-day.

Tongue red and raw-looking towards the tip. Another restless night was passed, and in the morning he looked thoroughly exhausted, although lying on his left side. Dullness with crepitations were well marked all over the right lung. The sputa were very tenacious, but untinged by blood.

The specific gravity of a sample of urine examined was 1,014. The pulse was 128, respiration 36. At about 1 p.m. the fever suddenly abated, and he broke out into a profuse sweating. At 2 p.m. he was given a scruple of quinine, in addition to the smaller doses of two grains which had been given every four hours from the commencement of his illness. At 6 p.m. his skin was cold and moist. Pulse 116, respiration 44. Soup was given every half-hour. His only complaint was of a severe pain along the outside of right thigh. At 10 p.m. the pulse had risen to 121, and the temperature to 102° 4.

During the night he vomited several times, and on the morning of the 15th, the seventh day of the fever, the pulse was 122, respiration 40. His tongue was dry, red, and rough. No vesicular murmur was heard at the base on right side, but the vocal thrill was increased. On the left side a few harsh bronchial râles were audible. He passed two stools this day, and in the evening his skin felt cool and moist, the pulse beating 112, and the respirations numbering 40. On the 16th his pulse was 125 in the morning, 132 in the evening; while at the former hour the respirations were 44, at the latter 48. Tenderness over cæcum was unmistakable. A pale, clay-coloured stool was passed at the hour of visit. The pain of the right thigh caused him much uneasiness.

Towards daybreak on the 17th the fever again lessened. The pulse was still 120, the respiration 48. In the evening there was another remarkable fall of temperature.

At sunrise on the 18th he was lying in a collapsed state, quite exhausted. The mouth was partially open, and the facial muscles kept twitching in a very peculiar manner. He was quite sensible, but restless, and tossing from one position to another. The pulse was 116, respiration 44, temperature 96° 7. Rum and soup were given more frequently, and in larger quantities than they had previously been. His features expressed pain when pressure was made over the right iliac region.

In the evening reaction had not come on. His skin was cold and clammy, and he lay with his legs drawn up, breathing 50 a

minute. The pulse was 124. Throughout the night of the 18th he was feverish and delirious, and in the morning he was lying on his back in a low typhoid state, with flies crawling unheeded over his face. The pulse was 128, respiration 48. When the right iliac region is pressed he calls out. He passed another clay-coloured stool this morning. At 3 P.M. hiccough began, and harassed him much. The tongue was moister in the evening, but its edges were red, as before. The pulse had fallen to 108, the respiration to 40.

On the morning of the 20th, the twelfth day of the fever, he was lying prostrate, with open mouth and half-closed eyelids, but quite sensible. The pulse was 124, the respiration 44. The spleen, which had been enlarged before this illness, was remarked this morning to have become much larger.

On the 21st he was lying with his legs drawn up, but whether there was tenderness of the abdomen or not could not be satisfactorily determined. There was no gurgling, but an expression of pain passed across his features when pressure was made. Twenty grains of quinine had been given last night, as the temperature fell. The pulse was 102 in the morning, 112 in the evening; the respirations being 34 and 40.

At sunrise on the 22nd he was lying collapsed with his knees drawn up and almost touching his chin. He calls out when pressure is made over the cæcum. Tongue dry and fissured. His pulse was 100, respiration 32. A red papula on the left side of chest was detected, but its exact nature was not ascertained. A bubo over the right parotid gland was very painful and caused much annoyance. Beseeches to be allowed to get up and bathe, and to have rice to eat. In the evening his hands were cold and clammy. Pulse 128, respiration 50. The parotid swelling was causing much pain, and the cæcal region was very tender.

At 3 A.M. on May 23, the fifteenth day of the fever, he died of exhaustion. Post-mortem examination four hours after death.

On opening the abdomen intense congestion of the peritoneal coat of the small intestines was observed. Until within two feet of the ileo-cæcal valve there was no ulceration of the mucous membrane, but immediately above it were two irregular ulcers, each an inch and a half long and one broad. Their long diameter was parallel with the axis of the bowel. Their edges were raised, tumid, and inverted. No tubercular or fibrinous

deposit was observed in the adjacent mucous membrane. Throughout the last two feet of the ileum were numerous small depressed ulcers, with an occasional patch of congestion. The mesenteric glands opposite the ulcers were enlarged and of a purple colour. When cut into, only a little red fluid exuded. No tubercular deposit here or in any other part of the body was found. In the descending colon a few cicatrices of old standing were seen.

Beneath both pleuræ were several patches of congestion. The base of the right lung was soft and œdematous, large quantities of frothy fluid pouring out when a section was made. The left lung was engorged throughout.

The liver was dark-coloured and unusually soft. The spleen was much enlarged, and of a dark venous colour. At the base of the brain there was considerable serous effusion. Over the right parotid gland suppuration had begun.

CASE LXXII.

ENTERIC FEVER.

(Dr. Wise, Dacca.)

Febrile temperature lasting to the fifty-second day. Sudden death on the fifty-sixth day, from asthenia.

Sheikh Yâsin, æt. 25, a cultivator from Cachar, was admitted into the Gaol Hospital, Dacca, on the morning of October 3, 1873. By his own account he had been taken ill on the 1st, with shivering and headache, followed by fever, which had never abated up to the time he came to the hospital. His temperature was then $101^{\circ}\cdot4$, his stomach was irritable, and bowels constipated.

Between October 3 and 7 the morning temperature was usually two degrees lower than the evening. The fever, in fact, was remittent in type. He complained of general pain throughout the body, of thirst, irritability of the stomach, and persistent headache. The belly was tender, and very sensitive to pressure. The least impact of wind made him shiver and roll himself in his blanket. The tip of the tongue was red and raw, the mucous papillæ being large and prominent.

A few bronchial râles were audible over the larger tubes on both sides. It was not until the seventh day that the characteristic pea-soup or yellow-ochre stools were passed. When tested they were invariably acid. The conjunctivæ became congested about the same date.

The urine was examined twice daily from the fifth day of the fever. In specific gravity it fell from 1,025 to 1,020 at the end of the first week. No albumen was detected. On the fifth and sixth days the chlorides diminished rapidly, and on the evening of the latter day they disappeared, and continued absent until the twenty-fourth day of the fever.

During the second week of the fever three or four loose stools were passed daily. They were either bright yellow or frothy, with green and yellow intermixed. The tongue was bright red, parched, and fissured. He generally lay on his back with the legs drawn up. The abdomen was always tender; and gurgling, not confined to the right iliac region however, was usually distinct. On the eighth day two red papules, fading on pressure, appeared over the left scapula and on the right breast. The pupils were natural, but the conjunctivæ were always injected. The skin remained dry and harsh. He slept tolerably well, and there was no delirium. Thirst was unquenchable. He took milk and soup readily, there being no gastric irritability. The pulse varied from 92, below which it never fell, to 110. The respirations when most rapid were only 24. The specific gravity of the urine fluctuated from 1,019 to 1,021. Chlorides were entirely absent. On the ninth day he complained of constant aching pains in his knees and calves. There was no swelling, or heat. It was not until convalescence was fairly established that these obscure pains left him.

During the third week the tongue gradually became moister, and on the sixteenth day the temperature fell to 98°·9. A scruple of quinine was given, but without any effect on the afternoon rise.

His motions were usually passed in bed. He lay, with his knees drawn up, sunk down in the bed. The tongue was often dry and hard in the afternoon. The knee joints were acutely painful, but no heat or swelling could be detected. The chlorides remained absent from the urine, which was of much lower density than during the previous week.

Between the twenty-second and twenty-eighth days of the fever the morning temperature approached the limits of health, but each afternoon there was an accession. He was very pale and bloodless, the extremities being dropsical. The diarrhœa gradually lessened, the tongue became clean and moist, and on *the twenty-fourth day chlorides again appeared in the urine.* On the mornings of the twenty-fifth and twenty-sixth days ten

grains of quinine were given to check the afternoon fever, but without effect. The urine varied from 1,017 to 1,029.

From this date convalescence began, although the afternoon rise of temperature still continued.

Quinine and other anti-periodics were given along with nourishing diet ; but it was not until November 21, the fifty-second day of the illness, that the fever entirely disappeared.

On November 23 he became jaundiced. He was then very weak, but able to move about and go to stool himself. During the night of November 27 he got up, stumbled in the dark, and fell. When raised up, he was dead. Post-mortem was held at 8 A.M. on November 28.

The body was greatly emaciated. The tissues generally were stained of a yellow colour. The brain was healthy, but very anæmic. The lungs were unusually pale ; otherwise quite normal. The heart was small. In both ventricles were several decolorised clots. The liver was of a deep yellow colour, weighing 2 lbs. 6 oz., and easily breaking down. The spleen only weighed 8 oz. The kidneys appeared healthy.

At the upper portion of the ileum were several enlarged Peyer's patches. The mucous membrane was pale, and the walls of the bowel were very thin, so that the swollen glands could be seen through them. The largest patch was about two inches long and half an inch broad. Its surface, swollen and mammillated, was of a pale pink colour. There was no ulceration remaining, and the edges of the patches were very little raised above the level of the adjacent mucous membrane.

CASE LXXIII.

ENTERIC FEVER WITH PNEUMONIA.

(Dr. Wise, Dacca.)

During the third week an eruption of rose-coloured spots. Hæmorrhage from the bowels on the sixteenth day. Recovery.

Rám Náth Chandál, æt. 25, was admitted into the Mitford Hospital on the morning of March 11, 1874. He had formerly been a domestic servant, but for some time he had been living unemployed in Islámpín, one of the central divisions of this city.

The account he gave of himself was to the following effect :— About March 1 he was taken ill with fever and diarrhœa, which have continued ever since. On the 5th he came to the hospital,

was admitted as an out-patient, and given medicine for intermittent fever. On the 8th he returned, and was ordered different medicine.

On the 11th he stated that the fever never left, but that at night it got worse, preventing sleep. He passed daily three or four thin, yellow stools; turmeric yellow was the term he used.

He walked up to hospital, and after resting some time he was examined. His pulse was 86, respiration 32; temperature $103^{\circ}5$. The eyeballs were sallow, the spleen enlarged; but no tenderness or tumidity of the belly was detected. The tongue was moist. On the centre and root was a thin, greyish fur, while at the tip the papillæ were enlarged and intensely congested. Moist râles, but no crepitations, were audible. On a careful examination of the chest, over the ensiform cartilage, a suspicious red spot was observed, which disappeared on pressure. Two grains of quinine with sulphuric ether were ordered to be taken every six hours.

March 12.—He passed two thin green and yellow stools during the night, which were very offensive. Pneumonia of the left lung was detected this morning. The urine was 1,018 in density. It contained no albumen, and the chlorides were scanty. The pulse was 80 in the morning, 84 in the evening; the respirations 36 and 40.

March 13.—He had a bad night, although twenty grains of chloral were given. Passed two thin yellow stools during the night. Dullness and crepitations audible over left side, and the expectoration is decidedly rusty. The red spot seen on the 11th has faded, leaving a faint brown discoloration. Over both shoulder-blades are several slightly raised papules, disappearing on pressure, and without any central punctation. The pulse was 80 in the morning, 86 in the evening; while at the same time the respirations were 36.

March 14.—Insists that he has no fever now. Has an anxious expression, but no stupor. Three stools, of same character as previous one, passed to-day. The spots on the back are very distinct and unmistakable.

This was the end of the second week of the fever.

March 15.—Two stools passed during the night were brownish-yellow, unctuous-looking, with white and dark particles floating in them. The spots on the back are convex, slightly raised, and paler than mosquito bites. Moreover, he

always wears a tight-fitting jacket impervious to mosquitoes. Urine 1,010, with excess of mucus; no albumen, chlorides abundant; amorphous lithates observed under the microscope.

March 16.—He passed two stools during the night, consisting of little but pure blood. At the bottom of the vessels were a few black clots, and upon the blood were floating a few bilious particles. He has no symptoms of scurvy, has never had piles, and the motions were passed without pain or straining. The tongue is showing a tendency to glaze, and the tip is still dotted over with bright red points. At 9 A.M. he passed a stool consisting of nothing but 'coffee-grounds,' very offensive, and without any clots. The belly was soft. No gurgling, tenderness, or even uneasiness could be elicited on pressure. Gallic acid with Dover's powder were given every four hours.

March 17.—A very foetid stool was passed during the night. It was liquid, brown, with here and there a greenish streak, and with small yellow bodies floating on its surface.

A careful examination was made by Surgeon J. Duke and myself of the eruption on various parts of the body. Powerful magnifying glasses were used. The spots marked on the 14th were still distinct, but losing their red colour.

There were peculiar difficulties in arriving at any positive conclusion regarding the spots, as they were interspersed with pimples of *acne punctata* and *coniformis*, and mosquito bites. The pale red, slightly-raised spots, fading under pressure, which appeared in successive crops, were determined to be the real spots of enteric fever.

March 19.—Two ochrey stools were passed during the night. In them were lumps of undigested casein, and numerous branny particles. No blood was observed. Abdomen soft and natural. After continuing uninterruptedly for eighteen days, the fever was gone this morning.

On the 20th and 21st from two to four thin ochrey stools were passed daily. On the first of these days the temperature was very low, $98^{\circ}\cdot1$; but this was partly due to his persisting in lying uncovered with a strong wind blowing over him.

On the morning of the 22nd he was sweating profusely. On the chest and epigastrium an abundant crop of sudamina was observed.

From this date the fever gradually abated. The morning temperature was below that of health, but the evening was at fever range. The diarrhoea was very obstinate, and continued

unchecked by astringents. Emaciation was well marked, and from being a strong, muscular young man, he was reduced to such a state of weakness that he required assistance to enable him to walk about.

He picked up strength slowly, but on April 3 he was discharged from hospital, and was able to walk to his home.

CASE LXXIV.

MALARIAL CACHEXIA WITH ENTERIC SYMPTOMS AND ULCERATION OF COLON AND RECTUM.

(Dr. Wise, Dacca.)

In the following case the cachexia, produced by long residence in an uncongenial climate by a native of the drier districts of Bihár, is well exemplified.

Dil Muhammad, æt. 25, a native of Mungir, was admitted into the Gaol Hospital on April 24, 1874, with fever and enteric symptoms. He was a thin, feeble man, pale and bloodless. The spleen was much enlarged, and he was subject to attacks of feverishness and diarrhœa, which, however, did not incapacitate him for performing his duties as a constable.

On admission his fever assumed a remittent type, and was accompanied by diarrhœa due to intestinal irritation or congestion, and by the discharge of pale urine of low specific gravity, without any traces of albumen. His stools were bright yellow, and numbered three to four in the twenty-four hours. His tongue was pale and flabby, his appetite was bad, and he had general heat and uneasiness throughout the abdomen. A few bronchitic râles were audible during his illness. He stayed in hospital till June 7, when he was discharged better in every respect. On June 24 he was readmitted, the fever having returned three days before. His tongue was red, bare of epithelium at the point, and with several longitudinal fissures furrowing its surface. His stools were passed several times a day, and resembled those of his former illness. The fever was not so strong as on the previous occasion, and the temperature varied from 101° in the morning to 103° in the evening. After taking a few doses of quinine the morning temperature fell to the natural range, but in the evening there was a slight febrile accession. On July 6 he was released from gaol, and nothing was seen of him till August 11, when he sought admission at the Mitford Hospital. He was then ill with diarrhœa due to muco-

enteritis, which had been getting worse for ten days. The stools were semi-fluid, of a yellow colour, and varied from three to nine in the twenty-four hours. There was usually no fever or abdominal pain, but now and then an evening accession of fever was noted. His legs and wrists were dotted over with an eruption of eczema vesiculosum. Medicines had little effect on the purging, and on September 2 his stools became foetid, the gums bled readily, the appetite was gone, and there was much pain and heat about the anus. These symptoms were unrelieved by medicines, and he died exhausted on September 3.

Post-mortem (8 A.M. September 4).—Body greatly emaciated. Both lungs were slightly congested behind, otherwise healthy. The bronchial glands were much enlarged, and filled internally with melanotic deposit. The heart was small, and destitute of fat. Its cavities were all healthy.

The right lobe of the liver was round and firm. When a section was made it was found to be of the 'nutmeg' character, the interlobular spaces being of a dirty white colour, while the centre of the lobule was intensely congested. Under the microscope an increased deposit of fat cells was observed in the midst of this grey substance.

The spleen was enormously enlarged, being over twelve inches in length. Its interior was soft and pulpy, and here and there clots of coagulated blood were observed.

Both kidneys were enlarged. The cortical portion was congested, especially around the bases of the cones.

The mucous membrane of the small intestines was generally tumefied and irregularly congested. In the ileum the patches of injection were most intense. No ulceration was observed. Peyer's patches were indistinct, and the solitary glands were not enlarged.

Throughout the large intestines the mucous membrane was congested, veined by large arborescent vessels, and discoloured by black pigmentary deposit. In the descending colon and rectum were numerous enlarged solitary glands surrounded by a halo of congestion. Several were crowned with a yellowish gritty deposit difficult of removal; while in the rectum this had been thrown off, leaving a small elevated circular ulcer with raised edges, the most advanced stage of chronic muco-enteritis (? dysentery).

CASE LXXV.

PNEUMONIA WITH ULCERATION OF SMALL INTESTINES.

(Dr. Wise, Dacca.)

Sheikh Sádha, æt. 32, was admitted into the Gaol Hospital on the evening of August 23, 1873, with fever. By his own account it had lasted continuously for eight days. He complained of thirst, headache, and nausea. His bowels were constipated.

On the morning of the 24th a purgative dose of 'Kála dána' was given. This was followed by eight stools.

On the 27th crepitation was detected over the bases of both lungs. The spleen was enlarged; urine 1,020, no albumen, chlorides scanty. Bowels relaxed; two stools were passed during night.

August 30, passed a restless night, delirious at times. Bowels again constipated. Urine 1,016; no chlorides, and no albumen. In morning pulse 114, respiration 28; in evening pulse 119, respiration 46. Tongue parched and becoming glazed.

September 1, pulse 118, respiration 38. Tongue dry and hard. Urine 1,020, traces of chlorides. In the evening he was lying on left side. His nights are passed in unrefreshing and broken sleep. Urine in evening 1,014. Mistura vini gallici at short intervals.

September 2, pulse weak and dicrotous. Slept better. Two bright yellow frothy stools passed during the night. Abdomen is tender on pressure.

September 5.—Hands very tremulous. Spleen much enlarged. A stool passed to-day consisted of blood chiefly. Tenderness in right iliac region is well marked. Pulse 112 in morning, 124 in evening. Respiration 42 and 46.

September 6.—Loud moist râles, with here and there fine crepitations, throughout both lungs. During night he passed four or five loose stools in bed. Urine 1,022, chlorides scanty.

On the morning of September 10, the nineteenth day in hospital, his temperature fell to 99°·2; but in the afternoon it rose to 103°·7. On September 13, the thirtieth day of the fever, there was complete abatement of abnormal temperature.

From this date convalescence appeared to be firmly established. On September 22, however, an afternoon accession of fever was noted. In the morning he was again cool. Inflam-

mation of the lungs was detected. After a few days all the symptoms improved, but only temporarily. He was very weak and greatly emaciated. His tongue became dry and cracked.

He passed frequent offensive stools in bed. On the morning of October 6 his pulse was 76, respiration 40. He was quite sensible, but in stupor. His eyelids were partially open, and his mouth wide. His right arm is firmly flexed. Several stools passed in bed. In evening he could not answer questions. Pulse 80, respiration 42. Right arm is still flexed. On the morning of October 7 he was found in a dying state.

Post-mortem (six hours after death).—The sinuses of dura mater were gorged with blood, and the arachnoid vessels were injected. No tubercular deposit was found in the lungs. The pleuræ on left side were thickened and inflamed, and the pulmonary tissue was congested. On right side the apex was oedematous, the base soft and engorged.

The liver, which was deeply congested, weighed 2 lbs. 7½ oz. The spleen was small, weighing 6 oz. The heart was healthy. Both kidneys were congested.

The mucous membrane of the stomach was swollen and pimples, with here and there points of extravasated blood. The duodenum was slightly injected; the jejunum was healthy. At the lower end of ileum were two ulcers, one circular, the other oval, both corresponding with the insertion of the mesentery. The edges of these ulcers were tumid and inverted. Their surfaces were covered with a thin transparent coating, beneath which red healthy granulations were sprouting. None of the solitary or aggregate glands were enlarged or otherwise altered. The mesenteric glands were enlarged and soft. Their interior was soft and spongy. No tubercular matter existed. The large intestines were quite healthy.

CASE LXXVI.

PNEUMONIA WITH ULCERATION OF GLANDS IN ILEUM.

(Dr. Wise, Dacca.)

Radha Náth Pal, æt. 42, was admitted into asylum January 13, 1862, with chronic mania. He was a shopkeeper by trade. In July he was in hospital with fever. On May 15, 1863, was a patient for diarrhœa and fever. On April 27, 1866, admitted with dysentery. In January 1867 had fever. In November symptoms of congested liver were detected. He was falling off in flesh,

and he had jaundice. He improved under treatment. At end of April 1868 he had an attack of remittent fever. In January 1869 he was again in hospital with jaundice. In August had another attack of fever. In June 1870 he was transferred to his father's care.

On June 18, 1873, he was readmitted. His health continued good until March 1874, but being a shy, reserved man he may not have been well for some time.

On March 1 he was admitted with fever. Pneumonia was detected after a few days. His pulse was dicrotous. On the second day his temperature was $104^{\circ}6$; on the third $104^{\circ}1$. Between the 11th and 14th days fever was moderate, but on the afternoon of the latter date temperature rose to $105^{\circ}3$. On the 15th and 16th it never fell below 104° . At 3 P.M. on the 15th it was $106^{\circ}2$; at 12 A.M. on the 17th $106^{\circ}1$. From that hour there was a rapid defervescence. During the night of the 18th he slept little, moaning and tossing about. In the morning respiration was 48, laboured. Does not speak to or recognise anyone. The pulse can be felt but not counted at the wrist. Tongue dry and brown. Is very restless, groaning continuously, and incessantly drawing towards himself the border of the covering blanket, or rubbing his lips with his hands. His eyes are bright and pupils natural. He is in a low delirious state, evidently sinking. He is lying on his right side with legs drawn up, abdomen being hard and unyielding. He died at 1 P.M.

Post-mortem (8 A.M., March 20, 1874).—Great serous congestion, and serous effusion beneath arachnoid.

Both lungs of a deep purple colour. Right intensely congested, soft and pulpy. Tubes filled with frothy mucus.

Left lower lobe in stage of grey hepatisation throughout; upper congested, oedematous and crepitant.

Large decolorised clots in both ventricles of heart extending into pulmonary vessels.

Liver gorged with blood, of a deep yellow colour. In gall bladder a large calculus weighing 54 grains.

Spleen not enlarged, but softer than natural.

Kidneys much enlarged, probably amyloid.

Numerous large patches of extravasation beneath mucous membrane of stomach, with large arborescent vessels, were found. The membrane appeared swollen, and mammillated in places.

The mucous coat in duodenum and jejunum injected here and there.

In ileum several large arborescent vessels with congestive patches were seen. At its lower end the interior of the bowel was variegated, pale, with small vessels branching over it in places. Opposite the insertion of the mesentery, one of Peyer's patches was swollen, and of a deep purple or blackish-blue tint. In the centre were two small ulcers almost healed. One, the size of a pin's head, was covered by a white film, with its edges granulating; the other, as large as a pea, had raised, very defined, and thickened granulating edges.

Near the latter was an enlarged, slightly prominent, indurated solitary gland, the size of a pea, which had ulcerated, and was now healing.

Two inches higher up was what appeared to have been a solitary gland, which had ulcerated and healed, leaving a white scar surrounded by a margin of dark colour like that seen after the falling off of a scab of small-pox.

The large intestines were slightly congested throughout.

CASE LXXVII.

ULCERATION OF SMALL INTESTINES IN A WOMAN DYING OF TUBERCULAR DISEASE OF LUNGS.

(Dr. Wise, Dacca.)

Abijha, æt. 35 years, a thin, cachectic woman, was admitted into hospital on April 6, 1874, with diarrhœa and tubercular disease of lungs. The looseness, which was generally clay-coloured, did not lessen under treatment, but went on increasing, and reducing her much. The left cheek on the 16th was œdematous, and the structures at the angle of the jaw became hard, painful, and red. By using poultices the swelling subsided, but it had not disappeared when she died of exhaustion at 6 P.M. on the 21st. There was no febrile temperature throughout her illness. The thermometer generally stood two or three degrees below the healthy range.

Post-mortem.—Abdomen, mesenteric glands were enlarged, and filled with tuberculous matter beginning to suppurate.

The duodenum, jejunum, and upper part of ileum were healthy. The last four feet of ileum were greatly congested, the mucous membrane being swollen and injected. In the upper two-thirds of this portion a few small pinhead-sized ulcers were visible. In the latter third innumerable ulcerated points, coalescing with one another, formed small ulcers, varying from a fourpenny-piece to a sixpence in size. Close to the cæcal

valve were two extensive ulcerated patches, which had apparently the same formation as the smaller ones. The solitary glands were distinctly enlarged, indurated, and filled with a yellowish-looking substance, probably tubercular.

The lower part of rectum was slightly congested, and showed the scars of former ulceration.

The apices of both lungs were disorganised and infiltrated with tubercular matter undergoing softening.

CASE LXXVIII.

CONTINUED FEVER.

(Dr. Manson, Amoy, China.)

Mr. B., about the beginning of June, had a sharp attack of ordinary summer diarrhoea. The violence of the attack abated, but for some time his bowels kept relaxed, and about June 10 he began to be feverish. By the 15th he was confined to bed, his temperature ranging from 103° to 104°·5. Fever and diarrhoea persisted for at least three weeks longer, and it was not until the 27th or 28th day of his illness that the temperature fell to normal. There was no marked iliac tenderness nor enlargement of the spleen, but he had delirium of a typhoid character, subsultus, and a few rose-coloured spots. At the outset he was liberally drugged with quinine without benefit; during the latter half of his illness the treatment was entirely expectant.

CASE LXXIX.

CONTINUED FEVER.

(Dr. Manson, Amoy, China.)

On June 18 or 19 Miss C. began to complain of langour, headache, pain in the limbs, and fever. She took quinine for several days without benefit. On the 7th day of her illness the thermometer had risen to 103°, and diarrhoea was frequent. Fever and diarrhoea continued for a week longer, and then gradually subsided; on the 21st or 22nd day she was convalescent. There were no spots, splenic enlargement, nor iliac tenderness.

CASE LXXX.

CONTINUED FEVER.

(Dr. Manson, Amoy, China.)

The patient had frequently had 'Tamsui fever.' On November 23 he felt pain in his left side; on the 24th was feverish; on the 25th had rigors, and was much prostrated, and he took some quinine; fever continued, and on the 28th he left Tamsui; on the 29th he arrived at Amoy. He had then considerable fever, prostration, pains in the neck, arms, and legs, and some tenderness of the epigastrium; his tongue was furred; he had severe headache, and he was covered from head to foot with an exanthem. The spots were circular, from $\frac{1}{16}$ " to $\frac{1}{8}$ " in diameter, red, not elevated, and disappeared on pressure. He had neither diarrhoea, iliac tenderness, nor enlargement of the spleen. The eruption kept on till December 4. During that night he perspired profusely, and on December 5 the eruption had entirely faded; this was the day of highest temperature. From that date the fever gradually subsided, and he was convalescent on the twentieth day of his illness.

CASE LXXXI.

CONTINUED FEVER.

(Dr. Manson, Amoy, China.)

A light-keeper was brought in this summer from Chapel Island, a bare rock, miles from any land or opportunity of infection, ill with a continued fever. I saw him on the seventh day of his illness. He had much headache, and was a good deal excited; but beyond the ordinary phenomena of simple continued fever, he had no particular symptoms. His temperature did not return to the normal point till the end of the third week. He took abundance of quinine, but apparently without any curative effect on the fever.

INSANITY AFTER MALARIAL FEVER.

I am indebted to Dr. Christie, of the Royal India Asylum at Ealing, for the following cases of insanity after, and apparently the direct result of, malarial fever:—

CASE LXXXII.

Gunner P. C., æt. 40 years, was received into hospital at Bellary, after an attack of severe intermittent fever, during which caries of the lower jaw resulted, destroying also the soft parts. The fever returned at times, and in ten months from the first attack a severe attack of mania came on; he was sent to England, and after about three years died. There did not appear to be any complication of intemperance or syphilis.

CASE LXXXIII.

Gunner T. B., æt. 20 years, admitted into hospital for intermittent fever, and discharged in about a fortnight. Re-admitted in three months with 'tinea capitis,' and again discharged in a month, but was then observed to be somewhat imbecile, and sent to the Lunatic Asylum at Bhowanipore, suffering from dementia. He was sent to England, and died from phthisis in about a year.

CASE LXXXIV.

Mr. T. W. H., a naval officer, attacked with intermittent fever in the Arracan Provinces; on its subsiding, he was sent to the sea, where he became the subject of acute mania, and was forwarded to hospital for treatment, but was sent to Bhowanipore. It appears he never was of a very strong mind, and some question of hereditary taint appears doubtful. He was sent to England, and improved considerably on the voyage, and after a month's residence in the asylum was discharged 'cured.'

CASE LXXXV.

Private R. E., æt. 23 years, admitted into hospital at Mooltan for repeated attacks of intermittent fever, attended with head affection; he was then observed to be very strange in manner, and was sent to the asylum at Colaba suffering from melancholia. After a year was sent to England and admitted into the asylum, from which he was discharged 'cured' in about twelve months.

CASE LXXXVI.

Gunner J. A., æt. 34 years, admitted into hospital for intermittent fever, and in a few days began to show symptoms

ILLUSTRATIONS.

of insanity by talking to himself. Sent to Bhowampore, and in about fifteen months to England, suffering from mania. Admitted to the asylum, and is still an inmate.

CASE LXXXVII.

Gunner W. B., æt. 35 years, admitted very frequently into hospital for intermittent fever, then took to drinking, and found his way to Bhowanipore. Sent to England, and admitted into the asylum for mania; he rapidly recovered, and was discharged cured in about six weeks.

These lectures were illustrated by maps showing the physical characters of India; the rainfall; isotherms and isobars; and the relative prevalence of malaria throughout the continent, and that of fevers among British troops in different stations in India. For these I am indebted to Dr. Sutherland and Mr. Trelawny Saunders. For the various statistical tables of the prevalence and effects of fevers and other diseases I am indebted to the Reports of the Sanitary Commissioners in India; to Dr. Sutherland, of the Army Sanitary Commission; and to Dr. Kynsey, P.M.D., of Ceylon. For several pathological specimens, illustrative of enteric fever and enlarged spleen, I am indebted to Professor Aitken, of Netley; and for some excellent drawings, by native artists, of the pathological changes in enteric fever, to Professor McConnell, of Calcutta. For all these, and for numerous observations and cases, I desire to record my sincere thanks to the gentlemen whose names are referred to in the lectures.

INDEX.

AFRICA (South), outbreak of enteric fever in, 210
 Albuminuria and malarial fever, case of, 138
 America, Drs. Drake, Dickson, and Woodward on the fevers of, 180
 Amoy, anomalous fever at, Dr. Manson on, 205
 — cases of continued fever at, 270
 Amritsar, outbreak of fever and cholera at, 62
 Anæmia from malarial fever, cases of, 138
 Animals, action of malaria on the lower, Profs. Axe and Fleming on, 47
 Aunesley, Dr., on fevers in India, 165, 166
 Ardent fever. *Vide* Thermal
 Army, the British, deaths in the, in India, 16
 Arnold, Mr. E. Lester, description of an attack of ague by, 72
 Ascension, enteric fever at, Staff-Surgeon Maclean on, 195
 Assam, prevalence of fever in, in 1880, 50
 — outbreak of enteric fever in, 211
 Asthenia, malarial cardiac, cases of, 133
 Asthma, malarial, case of, 134
 Axe, Professor, on the action of malaria on the lower animals, 47

B

BACILLUS MALARIÆ, researches of Profs. Klebs and Tominasi-Cru-delli on the, 27
 — Dr. Sternberg on the, 31
 Bellew, Dr., on the prevalence of fever in the Punjab, 54
 Bengal, mortality from fever in, in 1880, 17

Bermuda, enteric fever at, Dr. Don on, 198
 — Surgeon-Major Ferguson on, 268
 Bombay, mortality from fever in, in 1880, 17
 Bouchard, Prof., on typhoid as a specific miasmatic fever, 200
 Browne, Dr. B., of Lahore, on remittent and typhoid fever, 225
 Burtwan fever, Dr. Jackson's report on the, 183

C

CALCUTTA, mortality from fever at, 14
 — types of intermittent fever treated at the hospitals of, 69
 Ceylon, Dr. Kynsey on the fevers of, 201
 Chevers, Dr. Norman, on the prevalence of quotidian fever in India, 66
 — on typhoid fever in India, 173
 Cholera (and dysentery) relation of to fever, in India, 60
 — outbreak of, at Amritsar, 64
 Christie, Dr., cases of insanity after malarial fever, 271
 Cinchona alkaloids, employment of, in India, 113
 Clarke, Dr. Alfred, on typhoid fever in India, 177
 Climate of India, 4
 — great varieties of, 22
 Colin, Prof. Léon, on the non-specific character of typhoid fever, 182
 — on *typhoïde-palustre*, 186
 Coma, malarial, case of, 132
 Continued fevers in India, classification of, 153
 — cases of simple, 234, 245
 — at Amoy, Dr. Manson's cases of, 270

D

- DACCA, Dr. Wise**, on the fevers of, 189
Davidson, Dr., on enteric fever at Mauritius, 203
Diabetes in relation to malarial cachexia, 80
Dickson, Dr., on the fevers of America, 181
Don, Dr., on the climatic origin of enteric fever, 196
Drake, Dr., on the malarial fevers of America, 180
Duke, Dr., on the outbreak of fever at Amritsar, 62

E

- ECCLES, Dr.**, on fever in Thessaly, 52
 — on the effects of malarial fevers on wounds, 87
 — cases of remittent fever, 116
Embolism consequent on malarial poisoning, case of, 141
Enteric fever, account of, 164
 — first recognition of, in India, 166
 — etiology of, observations on the, 215
 — not traceable to filth-causes in India, 221
 — symptomatology of, 222
 — diagnosis of, 224
 — treatment of, 227
 — cases of, 247
 — with pneumonia, case of, 261

E

- EPHEMERAL FEVER** in India, 155
Febricula, in India, 155
 — cases of, 234
Ferguson, Surgeon-Major Dr. Johnston, on enteric fever at Bermuda, 208
Fergusson, Dr., on endemic fever on the shores of streams, 44
Fever, prevalence of, and mortality from, in India, 13
 — prevalence of, under different geological conditions, 44
 — distribution of, in India, 50
 — as affecting Europeans and Natives, 53
 — effects of season on the production of, 54
 — characteristics of, in India, 58
 — relation of, to cholera and dysentery, 60
 — diminution of, in India, 65

- Fever.** *Vide* Continued, Enteric, Ephemeral, Intermittent, Malaria, Remittent, and Thermic
Fleming, Prof., on the action of malaria on the lower animals, 47

- GANGRENE**, malarial, cases of, 142
Gordon, Dr., on typhoid fever in India, 173
Grabham, Dr., on enteric fever at Madeira, 207

H

- HEART**, malarial asthenia of the, cases of, 133
 fibrous concretion of, the case of, 143
Heat, fever from exposure to, 157
 — toleration of, in India, 160
 — effects of, on soldiers, 160
Vide Thermic Fever
Hemiplegia, malarial, case of, 135
Henderson, Dr., on organisms in malarial disease, 38
Hoystead, Dr., case of remittent with abscess of the liver, 229

- INDIA**, physical geography of, 3
 — climate of, 4, 22
 — temperature at various stations in, 6
 — rainfall in, 7
 — irrigation in, 8
 — population of, according to race, 10
 — habits and food in, 11
 — prevalence of fever in, 13
 — diminution of fever in, 65
Insanity from malarial cachexia, Dr. Christie's cases of, 271
Intermittent fever as it prevails in India, 65
 — condition of the urine during the paroxysm of, 71
 — Mr. Arnold's description of an attack of, 72
 — stages of described, 74
 — fatal collapse in, 76
 — production of chronic malarial poisoning by, 77
 — pathological anatomy of, 81
 — pigmentation and condition of the blood from, 84
 — cases of, 88
 — treatment of, 107
Irrigation in India, 8

- JACKSON, Dr., report of, on the
Burdwan fever, 183
Joubert, Dr., cases of remittent
fever, 127
— cases of continued fever, 245
— case of enteric fever, 255

K

- KLEBS, Prof., researches of, on the
bacillus malarie, 27
Kynsey, Dr., on fever, as observed
at Ceylon, 201

- LAVERAN, Dr., on the organisms of
malaria, 29
Liver, condition of, in intermittent
fever, 83, 85
— abscess of the, in malarial
fever, cases of, 229
London Hospital, diminution of
fever cases at the, 25
Lovell, Dr., on enteric fever at
Mauritius, 205

M

- McCONNELL, Prof., on typhoid and
remittent fever in Calcutta, 16
— on malarial pigmentation, 81
— on typhoid fever in India, 175
— cases of thermic fever, 241
MacCulloch, J. R., on the general
prevalence of malaria, 36
MacKenzie, Dr. Coull, on quotidian
fevers at Calcutta, 68
Maclean, Prof., on typhoid fever in
India, 173
— case of malarial fever with
abscess of the liver, 230
— Staff-Surgeon G., on enteric fever
at Ascension, 195
Macpherson, Dr. John, on typhoid
fever in India, 171
Madeira, enteric fever at, Dr. Grab-
ham on, 207
Malaria, nature of, 24
— decline of, in London, 25
— Dr. Salisbury on the germ-theory
of, 26
— the bacillus of, Profs. Klebs and
Tommasi-Crudelli on, 27, 32
— — Dr. Sternberg on, 31
— the organisms of Dr. Laveran, 29
— — Dr. Henderson on the, 38
— general prevalence of, MacCulloch
on the, 36
— drawing-room, 37

- Malaria, conditions favouring the
action of, 39
— prevalence of, under opposite
conditions, 45
— influence of subsoil water on, 45
— action of, on the lower animals,
46
— distribution of, in India, 49
— varying effects of, 51
— the diathesis produced by, 56
Malarial cachexia, production of, 70,
100
— plugging of the arteries of limbs
in, 79
— urethral fever in, 79
— diabetes in relation to, 79
— pigmentation of organs in, 81
— effects of, on the genital organs,
86
— treatment of, 115
— with abscess of liver, case of,
230
— with ulceration of the intestines,
Dr. Wise's case of, 264
Malarial fever, effects of, on wounds,
Dr. Eccles on, 87
— treatment of, 104
— bleeding in, Dr. Twining on,
105
— masked, 99
— — case of, 150
— insanity of, Dr. Christie's cases
of, 271
Manson, Dr., on anomalous fever at
Amoy, 205
— cases of continued fever, 270
Marston, Brigade-Surgeon G., on
enteric fever in India, 217
Martin, Sir R., on ulceration of in-
testines in fever, 169
— Surgeon-Major on enteric fever
in tropical regions, 200
Mauritius, enteric fever at, Dr.
Davidson and Mr. Lovell on, 203
Melancholia following malarial
fever, case of, 137
Moore, Deputy-Surgeon-General, on
the non-specific character of ma-
larial fevers, 34
— on the conditions favouring ma-
larial fever, 43
Morehead, Dr., on the varieties of
fever in India, 154
— on typhoid fever in India, 170

N

- NICHOLSON, Dr., cases of enteric
fever, 246, 252, 254, 255

North-West Provinces, deaths from fever in, in 1880, 19

O

OLDHAM, Dr., on typhoid fever in India, 178

Oude, deaths from fever in, in 1880, 19

P

PAROTIDITIS from malarial fever, cases of, 181

Pneumonia in enteric fever, Dr. Wise's cases of, 192, 261, 266

Population of India according to race, 10

Punjab, Dr. Bellew, on the prevalence of fever in the, 54

QUARTAN fever, cases of, 90

Quinine, administration of, 108

Quotidian fevers, great preponderance of, 66

— cases of, 88, 91

R

RAINFALL in India, 6

Remittent fever, characters of, 93

— description of the paroxysm of, 95

— complications of, 97

— pernicious forms of, 98

— masked, 99

— treatment, 108

— cases of, 116

— with abscess of the liver, cases of, 229, 230

— with enteric symptoms, case of, 246

Rheumatism with malarial fever, case of, 137

Ross, Dr., on the outbreak of fever at Amritsar, 62

Russell, Dr. E. G., on the effects of malarial disease on the spleen, 102

S

SALISBURY, Dr., on the organisms of malaria, 26

Smith, Deputy-Surgeon-General Alex., on typhoid fever in India, 174

Spleen, condition of the, in intermittent fever, 83, 85

Spleen, Dr. Russell, on the effects of malarial disease on the, 102

Sternberg, Dr., on the bacillus of malaria, 31

Sunstroke, effects of, on soldiers 160

Sutherland, Dr., on the factors in the production of malaria, 46

TEMPERATURES at various Indian stations, 6

Tertian fever, cases of, 90

Thermic fever in India, 156

— cases of, 236, 241

— with partial paralysis, case of, 238

— with nervous prostration, case of, 240

Tommasi-Crudelli, Prof., researches of, on the bacillus malarie, 27

Tubercular disease of the lungs with ulcer of the intestines, case of, 269

Twining, Dr., on bleeding in malarial fever, 104

— on ulceration of the intestines in fever, 168

Typho-malarial fever, Dr. Woodward on, 179, 181

Typhoid fever. *Vide* Enteric fever
Typhoide-palustre, Prof. Léon Colin on, 186

U

URETHRAL FEVER in relation to malarial cachexia, 79

— cases of, 143, 146

VERNEUIL, Prof., on glycosuria in relation to malarial cachexia, 80

W

WALL, Dr., on typhoid fever in India, 175

Whytlock, Dr., on typhoid fever in India, 178

Wise, Dr., on fevers in Dacca, 189

— on malarial pneumonia with enteric symptoms, 192

— cases of enteric fever, 256-269

Woodward, Dr., on typho-malarial fevers in America, 179, 181

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